A STUDY ON MICROALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS

DISSERTATION

SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE

M.D. GENERAL MEDICINE

BRANCH I

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI-600 032

APRIL 2017

CERTIFICATE

This is to certify that this dissertation entitled **"A study on microalbuminuria in acute ischemic stroke patients"** is a bonafide record of the work done by **Dr. Jayaram JK** under guidance and supervision in the Department of General Medicine during the period of his postgraduate study for **M.D General Medicine [Branch-I]** from 2014-2017.

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DECLARATION

In the following pages is presented a consolidated report of the study **"A study on microalbuminuria in acute ischemic stroke patients"** a cross sectional study, on cases studied and followed up by me at Sree Mookambika Institute of Medical Sciences, Kulasekharam from 2015-2016. This thesis is submitted to the Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of MD Degree examination in General Medicine.

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Dr. Jayaram J K

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ABBREVIATIONS

AHA American Heart Association

CBC Complete blood count

resonance angiography

CRP C-reactive protein

CT Computer tomography

CTA CT angiography

DUS Duplex ultrasound scan

DWI Diffusion weighted imaging

FDA Food and drug administration

ICA Internal carotid artery

IMT Intima media thickness

LACI Lacunar infarct

MCA Middle cerebral artery

MES Microembolic signals

MR Magnetic resonance

MRA MR angiography

MRI Magnetic resonance imaging

mRS Modified Rankin Scale

NIHSS National Institutes of Health Stroke Scale

NINDS National Institutes of Neurological Disorders and Stroke

OCSP Oxfordshire Community Stroke Project

PACI Partial anterior circulation infarct

POCI Posterior circulation infarct

SITS-MOST SITS-Monitoring Study

TACI Total anterior circulation infarct

TCD Transcranial Doppler

TCCD Transcranial colour coded Doppler

TEE Transoesophageal echocardiography

TIA Transient ischemic attack

TOF-MRA Time of flight magnectic resonance

angiography

TOAST Trial of Org 10172 in Acute Stroke Treatment

tPA Tissue plasminogen activator

WHO World Health Organisation

ABSTRACT

TITLE OF THE STUDY

A study on micro albuminuria in acute ischemic stroke patients.

BACKGROUND AND OBJECTIVES

Stroke is a medical emergency and can cause permanent neurological damage, complication and death if not promptly diagnosed and treated.

Micro albuminuria denote an abnormal increase in albumin excretion in urine. Albumin excretion range between 20-200 mcg/min or 30-299 mg/day. Role of micro albumin becomes apparent in acute diseases such as myocardial infarction and stroke.

Present study evaluate the incidence of micro albuminuria in ischemic stroke and its co relation to other risk factors.

METHODS

This study was started after approval of institutional ethical committee. This is a nonrandomized cross sectional study was done in medical wards and IMCU of the SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES, Kulasekharam. The study period was 18 months. During this period 50 patients were included. After meeting the eligibility criteria, a complete physical and systemic examination of the patients were done. Then investigations mentioned in the protocol was sent and results were analysed.

STATISTICAL ANALYSIS

The data was analysed by SPSS 16.0 with independent t-test.

RESULTS

In our study titled ‘A study on micro albuminuria in acute ischemic stroke patients’ a total number of 50 patients were studied. In this 25 were males and 25 where females. The average age group of the patients were 51 to 70 years. We found that the prevalence of micro albuminuria in patient with acute ischemic stroke was 74%. There was a strong possible relation of micro albuminuria as a risk factor for stroke.

MCA was the most common territory, measures 64%. Facial nerve was the most common cranial nerve involved with 64% of total case. 62% had ECG showing LVH pattern and there was strong correlation with micro albuminuria.

Positive correlation between lipid profile and micro albuminuria in stroke patient.

Significant correlation between blood sugar and blood pressure with microalbuminuria in acute ischemic stroke patients.

CONCLUSION

In our study we have found out that there is a statistically significant association between microalbuminuria and incidence of acute ischemic stroke.

SUMMARY

We conducted a study titled “A STUDY ON MICROALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS” which was done as a non-randomized cross sectional study in the medicine wards and IMCU of Sree Mookambika Institute of Medical Sciences, Kulasekharam for 18 months study period on 50 ischemic stroke patients admitted and met pre-defined criteria gave informed consent after obtaining ethical clearance from the institutions ethical clearance committee.

In our study of 50 patients with ischaemic stroke, 74% had significant microalbuminuria. MCA was the most common territory involved with 64% cases followed by PCA in 10 % and ACA IN 8 %. But no significant correlation of microalbuminuria with particular vascular territory has found out. In our study 62 % cases had ECG showing LVH pattern. crainal nerve involvement most commonly facial nerve was seen in 74% of all cases. There was a positive co-relation between lipid profile and LVH with microalbuminuria . There is statistically significant co-relation between elevated blood sugar and elevated blood pressure in patients admitted with ischemic stroke less than 24hours. There was strong positive relationship of microalbuminuria in acute ischemic stroke patients and microalbuminuria is an important independent risk factor for stroke.

INTRODUCTION

Stroke (cerebrovascular disease) is due to disturbance in the blood supply to the brain which causes rapid loss of brain function[1]. According to WHO stroke is defined as `Rapid development of clinical signs and symptoms of focal neurological disturbance lasting more than 24hrs. or leading to death with no apparent causes other than vascular origin.[2]

Stroke can cause a permanent neurological damage so it has to be diagnosed and treated as a medical emergency. It is one of the leading causes of serious disability[3].

There are two types in Stroke 1. Ischemic and

2. Hemorrhagic stroke.

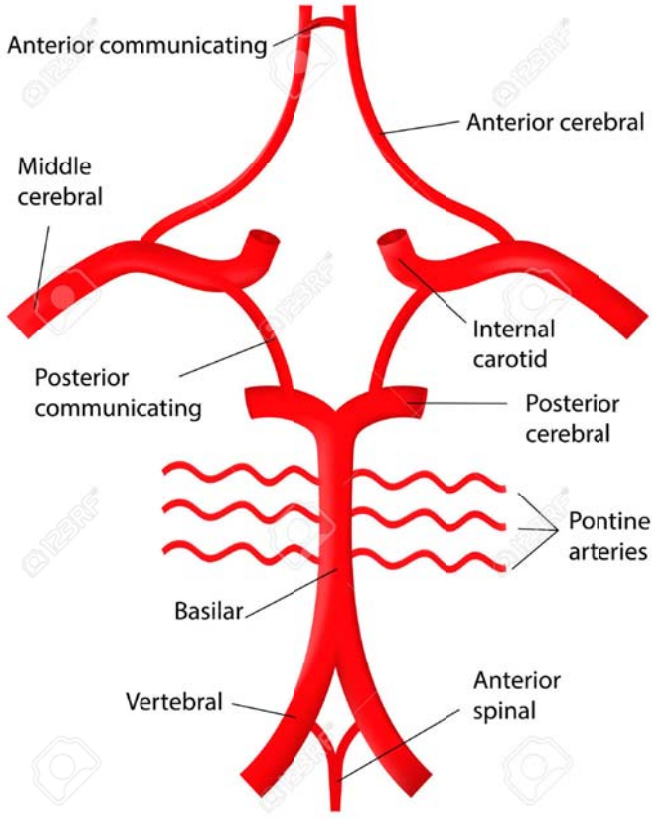


Figure 1: Circle of Willis

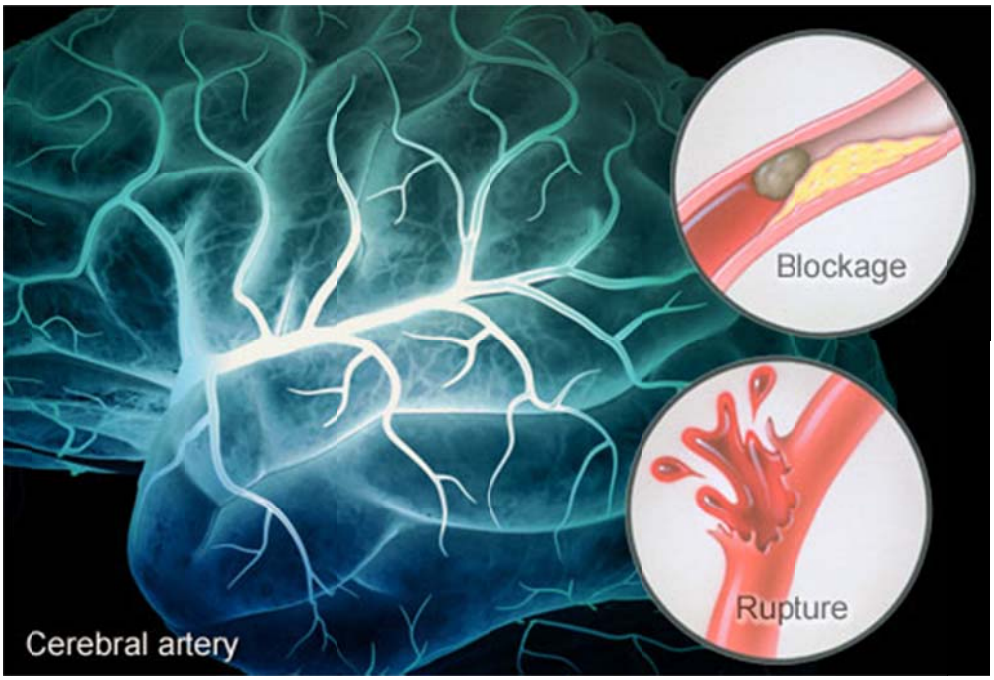


Figure 2: Types of stroke – Ischemic(blockage) and Hemorrhagic(rupture)

Ischemic stroke is due to sudden occlusion by a thrombus or an emboli in the artery to brain. 50-85% of all stroke are ischemic stroke[4].

The prevalence rate of stroke is about 84 to 262/1,00,000 in rural areas and it is about 334 to 424 in urban areas.[5] Recent studies conducted in India estimated that the prevalence rate increased from 0.1to 0.3 /1000 in the less than 45years of age group to 12-20/1000 in 75-84 years age group. Stroke prevalence in rural places of India was 1.1% and in urban area India was 1.9%. Prevalance of stroke is inversely proportional to the level of education. It is directly proportional to the age of the population.

Following are the main risk factors of stroke in India. Though there are no sufficient amount of data available about the co-existent risk factors in India and other south Asian countries, the following 3 transitions(demographic, socioeconomic, life style) which increase both the modifiable and non-modifiable risk factors of stroke.[6-7] For example lifestyle modifications like reduced physical activity, increased food intake and demographic changes like increase life expectancy are the reasons of increased prevalence of risk factors of stroke. Age, sex, low birth weight, ethnicity and genetic factors are the important non modifiable risk factors.

Patients with hypertension and diabetes are already at a risk for cerebro-vascular events, in addition to this microalbuminuria is studied to be an independent risk factor for stroke.

The correlation of microalbuminuria with other risk factors of stroke such as diabetes and hypertension has been studied in the past, and most studies show a very strong association in predicting stroke and also as a predictor of mortality post stroke.

As per the study in Gujarat, the commonest causes of stroke in elderly people are hypertension(40%), alcoholism(35%), smoking(23%) and hyperlipidemia(17%). In young people, the common causes are smoking, alcoholism, increased BMI, diabetes and hypertension. The risk factor for atherosclerosis in large vessel and occlusion of small vessel are hypertension and diabetes.

Microalbuminuria denotes an abnormal increase in albumin excretion in urine below the lower limits of sensitivity for routine diagnostic methods.[8]

The current National Kidney Foundation's definition of microalbuminuria is excretion rate of Urinary Albumin is between 20-200 microgram/min or 30-299mg/day[9], which was used as a marker for incipient diabetic nephropathy.

In past decade the role of microalbuminuria has become apparent in Myocardial Infarction and stroke.[10]

Microalbuminuria indicates irregularity of the microvasculature of the kidney. It also indicates the global endothelial dysfunction of kidney that is associated with increased risk of cerebrovascular events like stroke[11-12]. The current proposed patho physiological mechanism is through local vascular smooth muscle and endothelial cells injury due to vascular shear stress, changes in Nitric Oxide and increase in cytokines and increased vascular permeability. Other studies have also shown that the presence of microalbuminuria post stroke or episodes of transient ischemic attacks is also a very strong indicator of recurrence of stroke with or without other risk factors for stroke. It is also said that microalbuminuria is associated with vascular disorder of heart and kidney similar to those of cerebrovascular disease.

Estimating microalbuminuria is a simple noninvasive dip stick procedure, which opens to the window for prediction of several vascular events and its association with various other major risk factors.

AIMS AND OBJECTIVES

The aims and objectives of this study were :-

- To know the incidence of microalbuminuria in acute ischemic stroke patients (i.e less than 24 hours).
- Role of microalbuminuria as a risk factor in stroke.
- Prevalence of microalbuminuria in different subtypes of ischemic strokes (MCA/ ACA/ PCA)

HYPOTHESIS AND SCIENTIFIC JUSTIFICATION

Scientific Justification of the study:

One of the leading causes of diability and death is stroke. It is mainly due to the increased prevalence of modifiable risk factors, demographic changes, doubling the burden of non-communicable diseases. The impact of stroke is more in poor people due to change in prevalence of risk factors and inability to afford the high cost stroke treatment.

Most of the stroke survivors live with disability so that family members have to spend lots of money for rehabilitation which leads to poverty in the family.

The role of risk factors such as hypertension, smoking, diabetes, dyslipidemia in the cause of stroke has been in substantial progress. However, uncertainty remains regarding the significance of more novel risk factors in contributing to the burden of stroke. In particular, several prospective studies have suggested that the presence of protein in urine is associated directly with cardiovascular events, including stroke[14-20]. Better understanding of the precise nature of the relationship between proteinuria and stroke is important from both a clinical and public health perspective.

Even though microalbuminuria is associated with diabetes, hypertension, ageing, history of myocardial infarction, left ventricular hypertrophy which are the risk factors for stroke, there is only little information about microalbuminuria as a predictor or independent risk factor of stroke[21].

Over the last 4 decades, several prospective clinical studies have identified a series of independent risk factors for symptomatic vascular events including stroke and also showed a better understanding of the multifactorial pathogenesis of atherosclerosis. The underlying entity behind most vascular events and the fact that many of these event occur in persons who do not harbor convusional vascular risk factors has promoted to search novel risk factors for prediction of stroke.

The clinical value of many of these emergency risk factors remain uncertain. One such emerging vascular risk factor is microalbuminuria. Microalbuminuria is an independent risk factor for stroke and also interacts with several vascular risk factors.

Though there is convincing evidence of an independent positive relationship between overt proteinuria and stroke risk, but there is no investigations done to know the relationship between microalbuminuria and stroke incidence. In this study, our aim is to know the strength and consistency of association between microalbuminuria and the incidence of ischemic stroke.

REVIEW OF LITERATURE

Stroke definition

As per the definition by WHO stroke as a "neurological deficit of cerebrovascular cause that persists beyond 24hours [1]

Stroke Types[2]

Stroke is classified

Based on timing

- Primary
- Secondary/ recurrent

Based on etiology

- **Ischemic stroke -** about 83% of all stroke cases are ischemic stroke. It is due to obstruction of blood vessels supplying brain.
- **Hemorrhagic stroke** occurs when there is rupture of a vessel or intracranial bleed.

Based on how far it develops

- Acute
- Chronic

Risk Factors for Stroke [3]

Risk factors for stroke are non-modifiable and modifiable risk factors

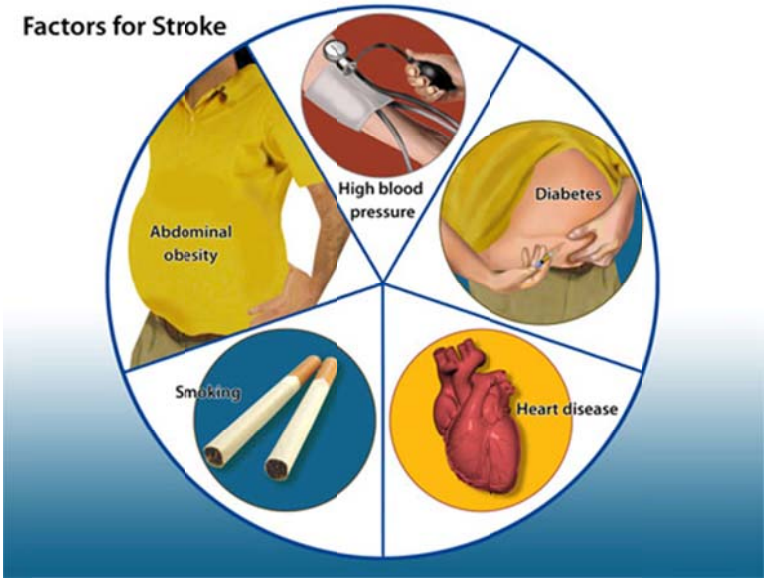


Figure 3: Modifiable risk factors for stroke

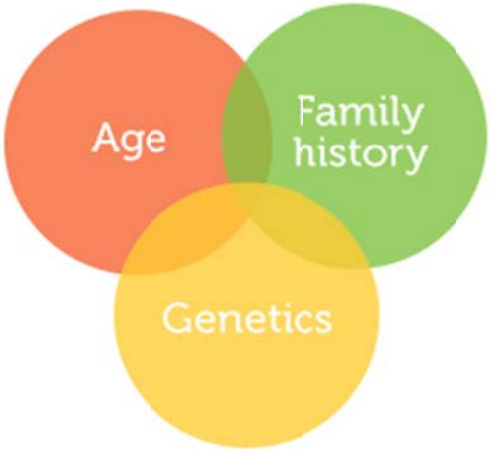


Figure 4: Non modifiable risk factors for stroke

Modifiable Lifestyle Risk Factors

Cigarette Smoking[4]

Promotes stroke by the following factors.

- Distensibility/compliance of blood vessel is reduced,
- Fibrinogen levels increased,
- Platelet aggregation increased,
- HDL cholesterol levels decreased, and
- Increased hematocrit
- 18% of stroke patients give a history of active cigarette smoking. Not only that, even passive cigarette smoking increase the risk of stroke by causing atherosclerosis progression.

Alcohol Consumption [5]

Heavy alcohol consumption favors stroke by

- Elevating blood pressure, increasing the coagulability
- Enhancing the occurrence of cardiac arrhythmias, and
- Decreasing the cerebral blood flow

Sedentary life style Increased physical activity reduce the pro-athrogenic factors. As per the meta-analysis study done to know the relationship of physical activity with stroke risk, more active subjects had lower risk of stroke or mortality when compared to less active subjects.

Non-Modfiable Risk Factors

Age as a predictor for stroke

An important non modifiable risk factor of stroke is age. The risk of stroke incidence doubles per decade after 55 years of age[6].

Stroke are rare in children most often due to hemorrhage.

In India stroke occurs at a younger age than other countries, which is 15 years younger than other well developed countries.

Gender as a predictor for stroke [7]

Males are more prone for development of stroke than females , but the severity of stroke is more in females.

Race and ethnicity

Geography

Hereditary

The potential new risk factors

Genotypes,

Inflammatory markers [8,9]

The potential predictors of risk of stroke and prognosis are inflammatory biomarkers like high sensitivity C-reactive protein and lipoprotein associated phospholipase A_2. Stroke can also be precipitated by infection. Independent of lipid lowering effects in the drugs like statins decrease the levels of inflammatory biomarkers. The ability of stating in reducing the stroke and coronary events has correlation with effect of statins on inflammatory biomarkers.

Functional markers[10]

- **Microalbuminuria**

The increased thickness of tunica intima-media of common carotid artery is a result of microalbuminuria and atherosclerosis found in endothelial dysfunction. The most important screening test for finding the risk of atherosclerotic disease is the measurement of urinary microalbumin. It is also used to determine the people who are prone for developing ischemic stroke.

Etiology of ischemic stroke

JP Mohr - 1997studied the various etiologies of stroke due to ischemia[11]

- **Embolism Cardiac or Aortic Origin migrating to brain**
  - **Myocardial Infarction**[12]

One of the important complication of anterior wall MI is stroke. It occurs in about 1 to 3% of all infarcts and in about 2 to 6% of patients with anterior wall MI. According to ECG studies in about 40% of anterior wall MI patients, left ventricular mural thrombosis occurs. Atrial fibrillation after AMI is an independent risk factor for stroke.

  - **Atrial Fibrillation**[13]

Cerebral infarction is as a result from embolization of intracardiac thrombi, mostly from the left atrial appendage. The risk of recurrent stroke is very high in the first two weeks after the myocardial infarction.

- **Disease of the heart valves**[14]

Ischemic stroke and cardiac valve disease has known association. Because embolism formed in native valves or prosthetic valves migrates and causes occlusion of blood vessels supplying brain.

- **Native Valves**[1]

Most common cause of thromboembolism is Rheumatic mitral stenosis.

- ***Mitral Valve*** **Prolapse**[16]

Fibrinous deposits and endothelial denudation are seen in mitral valve prolapse. Annular thrombus at the junction with atrial wall is also seen in Mitral valve prolapse. Thromboembolic events are more formed in myxomatous and redundant valve leaflets.

Mitral stenosis and regurgitation, cardiac arrhythmias, abnormality in conduction and cardiogenic brain embolism are associated with calcifications of mitral annulus.

- ***Cardiac Prosthetic Valves***[17]

Tissue prosthetic valves has decreased risk of production thromboembolism when compared to the mechanical valves

- ***Repaired Heart Valves***[18]

Percutaneous balloon valvuloplasty of mitral valve is associated with risk of embolization, the risk of stroke is approximately 2%.

- **Aortic Arch Origin[19] of emboli**

Atherothrombotic or cholesterol embolism formed as a result of complicated atherosclerotic plaques in the aortic arch.

- **Patent Foramen Ovale[20]**

The patent foramen ovale is a risk factor for stroke patient shown by de Belderet al .

- **Ischemia of brain due to reduced perfusion and Artery-to-Artery Embolism[21-**

Severe stenosis of carotid and basilar artery causes reduced perfusion leading to stroke. Micro stenosis of deep arteries also causes reduced perfusion. The most distal territories are affected prior to the most proximal territories which is termedas border zone(watershed infarction).

- **Atherosclerotic Plaque in large arteries.**

The most common cause of cerebral infarction associated with large cerebral artery plaques is artery to artery embolism.

- **Vasculitis**[24,26]

Inflammatory conditions like granulomatous angiitis, Giant cell arteritis, SLE(systemic lupus erythematosus), and polyarteritis nodosa are the conditions come under vasculitis.

The stroke mechanism alters like necrotizing vasculitis, hypercoagulable state, artery-to-artery or cardiac embolism. In the arteria, inflammatory infiltration is seen in bacterial or tubercular meningitis paient, herpes zoster, arteritis, cysticercosis of brain and fungal infection.

- **Other arterial diseases**

- **Occlusion**[24] **of small arteries.**

The small artery occlusion cause small ,deep lacunar or cavitory infarcts which forms the basic principle behind the small deep infarcts. The small arterial narrowing can cause focal zones of ischemic rarefaction –non cavitary , infarct of white matter or incomplete-which are not true infarcts by histology

- **Intrinsic Small Artery Disease**

The most common pathology in symptomatic lacunar infarcts is micro atheroma. In hypertensive patients the occlusion small penetrating arteries(less than 200 μm) is due to lipohyallinosis or segmental small arterial disorganization. This also leads to small asymptomatic lacunar infarcts.

Ischemic rarefaction and small deep cavitory infarcts are due to small granular arteriopathy associated with cerebral autosomal arteriopathy with subcortical infarct and leucoencephalopathy [11]

- **Other Causes of occlusion of Small Vessel**

The potential cause lacunar infarction is cardiogenic embolism. The first indication of critical large intracranial artery stenosis is lacunar syndrome.

In the upper lateral borders of lateral ventricles, small deep infarcts are seen along with ipsilateral carotid occlusion in a deep watershed territory called as low flow or nternal water shed infarcts.

Hemodynamically significant cardiac disease other vasculitides and prothrombotic conditions are risk factors for stroke

- **Prothrombotic States[24]**

Primary prothrombotic states

- Antithrombins, heparin cofactor II,
- Proteins C and S, and
- Derangements of Fibrinolytic system.
- Antiphospholipid antibodies

Stroke Classifications

TOAST Classification[27]

Trial of Org 10172 in Acute Stroke Treatment (TOAST classification use most commonly to subtype ischemic stroke .

TOAST classification categorize ischemic stroke based on etio-pathology.

Etio-pathology of stroke was determined based on clinical examination, neuroimaging with CT or MRI, vascular imaging using angiography or ultrasound and cardiac investigations.

Ischemic stroke patients are classified into following subgroups:

1) Atherosclerosis of large arteries.

2) Cardio embolism,

3) Occlusion of small vessels,

4) Stroke of other determined etiology,

5) Stroke of undetermined etiology,

OCSP Classification

OCSP classification describes the anatomical localization of infarct based on clinical subgroups [28,29]

Ischemic stroke is classified into following sub-groups:

1) Total anterior circulation infarct (TACI),

2) Partial anterior circulation infarct (PACI),

3) Posterior cerebral infarct (POCI) and

4) Lacunar infarct (LACI).

The risk of stroke is double in patients with coronary artery disease.[30] The attributable risk of stroke is about 12%.

Diabetes[31]

The risk of stroke is independently related to diabetes.

Dyslipidemia[32]

The ischemic stroke is associated with increased serum triglycerides, total cholesterol and LDL cholesterol.

Prognosis in Stroke Outcomes

Stroke is the leading of long-term disability. The case fatality rate in hemorrhagic stroke (33–45%) is higher than ischemic stroke (8–12%). Men have higher age adjusted stroke mortality rates comparing to that of females. [33]

Clinical Diagnosis

Clinical symptoms of ischemic stroke depend on the anatomical location of the thrombus. Stroke usually presents with an acute loss of brain functions. These functions usually involve the realm of motor, sensory, language, vision, visuo-spatialperception or consciousness.

A transient ischaemic attack, or TIA also known as “mini-stroke” or “warning stroke” in which symptoms of stroke are resolved within 24 hours.[34]

ABCD2 score is useful in predicting the short-term stroke risk after a TIA[35]:

A = Age

B = Blood pressure

C = Clinical symptoms

D = Duration of symptoms

D = Diabetes.

ABCD2 after a TIA

Age 60>	Yes	+1
BP > 140/90 mmHg at initial evaluation	Yes	+1
Clinical features of TIA:		
Unilateral weakness		+2
Speech disturbance without weakness		+1
Duration of symptoms?	10-59mins	+1
	Over 60 mins	+2
Diabetes mellitus in patient's history?	Yes	+1

HIGH RISK – score of 4 or above. Should be consulted within 24 hours in a specialist TIA clinic.

LOW RISK – any score of 3 or under 3. Should be consulted within 7 days in a specialist TIA clinic.

Most ischemic strokes presents as a sudden loss of function in one of the domains.[36] However, a sudden loss of neurological functions in the above domains could represent pathologies other than ischemic stroke. These include intracranial hemorrhage, seizures, vasovagal syncope, migraine, tumor, meningitis etc. Clinical examination along with neuroimaging secures a proper diagnosis of ischemic stroke.

SYNDROME	ANATOMY INVOLVED	MAJOR SYMPTOMS	VESSELS INVOLVED	ETIOLOGY
Left MCA	Left frontal/parietal cortex and subcortical structures	Aphasia, right visual field cut, right motor/sensory deficits; face > arm > leg weakness; left gaze preference	Left MCA or major branch; could also be left ICA or siphon	Emboli from heart or proximal lesion; intrinsic atherothrombosis
Right MCA	Right frontal/parietal cortex and subcortical structures	Neglect syndrome, agnosia, apraxia, left motor/sensory deficits, visual field deficit; right gaze preference	Right MCA or major branch; right ICA or siphon	Same as left MCA
Left ACA	Left frontal and parasagittal areas	Speech disturbance, behavioral changes, leg > arm weakness	Left ACA	Intrinsic atherothrombosis, embolic
Right ACA	Right frontal and parasagittal areas	Behavioral changes, leg > arm weakness	Right ACA	Same as left ACA
Brainstem	Pons, midbrain, medulla, cerebellum	Ophthalmoplegia, bilateral motor deficits, ataxia/dysmetria; nausea/vomiting/vertigo, coma/altered mentation	Basilar artery	Intrinsic atherothrombosis, embolism from heart or proximal vessel
PCA	Upper midbrain, occipital cortex/subcortex, thalamus, medial temporal lobes	Visual field cut, motor/sensory loss, seizures, gaze problems; 3rd nerve deficits	Posterior cerebral artery, thalamic perforators	Embolism from proximal lesion intrinsic atherothrombosis

ACA, anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery.

Figure 5: Function loss and domain relationship

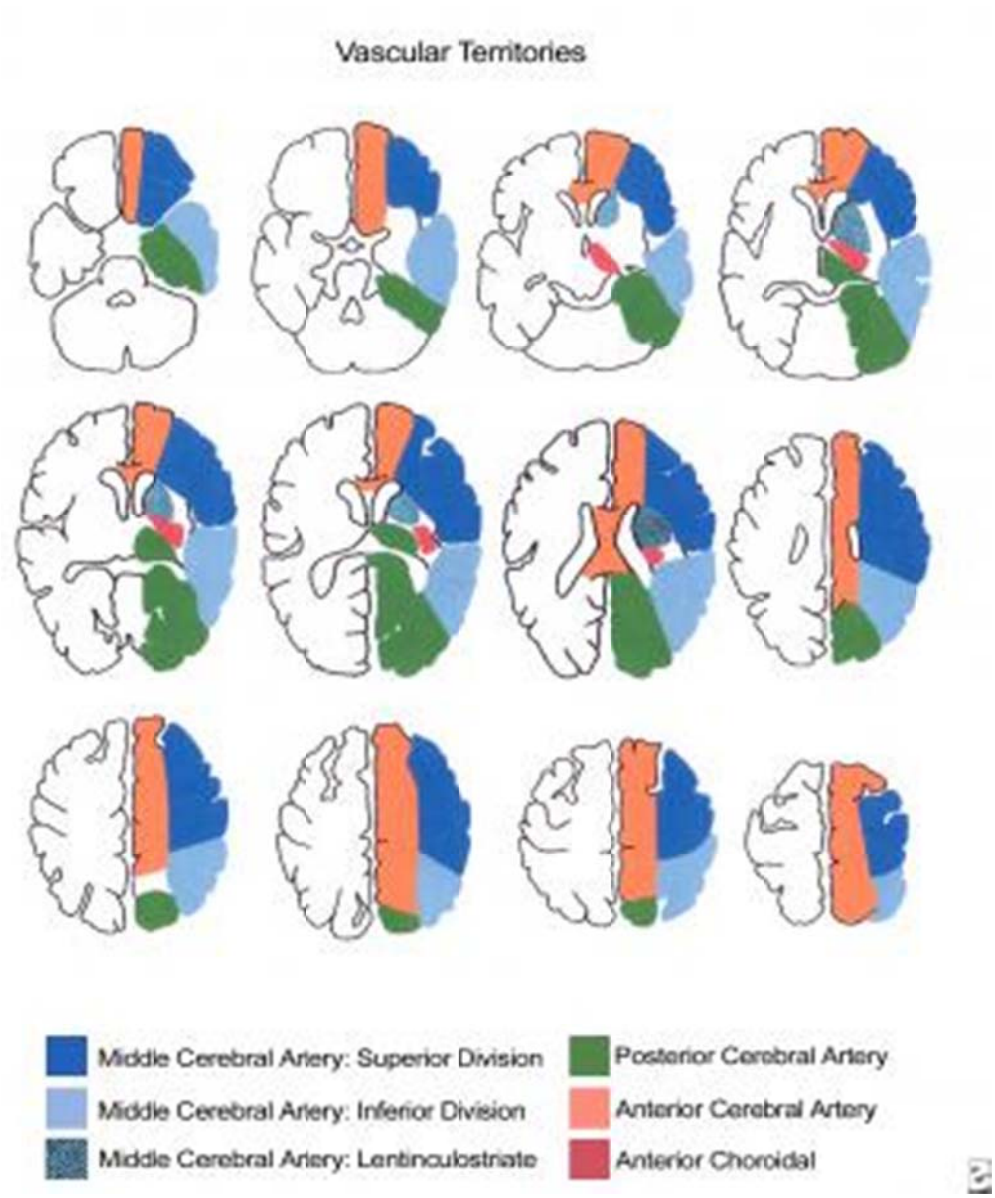


Figure 6: vascular territories

Treatment of acute ischemic stroke

Stroke treatment was revolutionized with the introduction of tissue plasminogen activator for intravenous thrombolysis. [36]

Supportive therapy for acute ischemic stroke

Supportive measures are very important in the acute phase factors such as high body temperature and high blood glucose may have deleterious effect on stroke outcome [37,38]

Neuroprotection

Neuroprotection refers to the process of protecting neurons from apoptosis and ischemic degeneration. At present no neuroprotective agents are proven to have a definite effect in stroke patients.

Components of stroke care unit.[39]

In India implementation of stroke care unit is a big challenge and at present only 35 stroke units are available. Most of them were under the private sector.

Thrombolysis

Thrombolysis of stroke is a common practice in India.

Rehabilitation

Physiotherapists and speech therapists in India are mainly concerned with rehabilitation.

Drugs for secondary prevention

Antiplatelet drugs, lipid lowering drugs should be initiated early for secondary stroke prevention.[40]

Complications of stroke [41-46]

P Langhorne et.al in 2000in their study found the medical complications were as follows: [41]

- Neurological—
  - Recurrent stroke (9% of patients),
  - Epileptic seizure (3%);
- Infections—[42,43]
  - Urinary tract infection (24%),
  - Chest infection (22%), [43]
  - Others (19%);
- Mobility related—
  - Falls (25%),
  - Falls with serious injury (5%),
  - Pressure sores (21%); [44]
- Thromboembolism—
  - Deep venous thrombosis (2%), [46]
  - Pulmonary embolism (1%);
- Pain—

- Shoulder pain (9%),
- Other pain (34%); and

- Psychological—

- Depression (16%), [50]
- Anxiety (14%),
- Emotionalism (12%), and
- Confusion (56%).

Prognosis in stroke

Ischemic stroke subtypes have better prognosis as compared to stroke of cardio embolic stroke [51-53]

M Prencipe - 1998 [54]studied the minor ischemic strokes with minor or no disability by Rankin score and found that the 10-year mortality rate was 32%, with a relative risk of 1.7 (95% CI, 1.4 to 2.1) compared with the age- and sex-matched general population. Age, minor disability, myocardial infarction, non valvular atrial fibrillation and hypercholesterolemia increase the risk of disability and death, recurrent minor strokes.

Hypertension has an association with death and stroke recurrence[55]. Stroke recurrence is mainly predicted by the presence of hypertension in the Rochester study.[56]

MICROALBUMINURIA

The mechanism of renal filtration of albumin

Lazzara and Deen in 2007[57]showed that through glomerular filtration membrane, albumin and protein reabsorbed in proximal convoluted tubule by receptor-mediated endocytosis.

Birn et al. [58]called the receptor complex called megalin-cubilin is involved in the endocytosis process. Albumin then undergoes degradation in the lysosome into amino acids which return to the blood stream.

Microalbuminuria –definition [59]

30 - 300 mg per day level of albumin excreted is microalbuminuria (equivalent to 20 to 200 µg/minute in a timed overnight urine collection, 20-200 mg/L on spot urine specimen or ACR 2.5 to 25 mg/mmol in males or 3.5 to 25 mg/mmol in females)

In **PREVEND study**[60] prevalence of microalbuminuria remains fairly constant for females, despite of their age group, but in males prevalence increases after the age of 50yrs. In our study prevalence of microalbuminuria was higher in younger males as compared to older males(66.66% in males 50yrs of age). In the present study, the difference in occurrence of microalbuminuria were significant.

Category	Spot Collection	Timed Collection	24-hr Collection
Normal	Less than 30 mcg/mg creatinine	20 mcg/min	30-300 mcg/mg creatinine
Microalbuminuria	30-300 mcg/mg creatinine	20-200 mcg/min	30-300 mg
Clinical albuminuria	More than 300 mcg/mg creatinine	More than 200 mcg/min	More than 300 mg

Table 1. American Diabetic Association Classification of Microalbuminuria[61]

The various mechanisms underlying have been hypothesized

In a study by **Satchell** [62] they reported that alteration of renal haemodynamics and endothelial changes is the mechanism underlying microalbuminuria that This process is also regulated by feedback mechanism of the afferent arteriole proposed by **Helal** [63] in 2012 and is mediated by specific cells in the distal convoluted tubule when the volume of filtrate is high resulting in constriction of afferent arteriole to maintain GFR as shown by **Mountokalakis**, in 1997 [64].

DallaVestra et al.[65], 2003 said that the mechanism underlying microalbuminuriais was probably due to impairment of glomerular permeability.

The various mechanisms underlying microalbuminuria proposed is the

- Structural changes that allow passage of albumin in the urine in large quantities
- Podocyte impairment
- Impairment in the glycocalyx
- Functional impairment of the renin-angiotensin aldosterone system

  Angiotensin II is associated with increase in production of inflammatory mediators and reactive oxygen species which leads to sclerosis of podocytes.
- Generalized endothelial dysfunction
- Changes in the endothelial production of nitric
- Renal hemodynamic abnormalities as a result of increased blood pressure may increase glomerular permeability.

Microalbuminuria and disease

In the recent time micro albuminemia has been proven to be associated with various diseases.

- **Garg and Bakris[66]** in their study microalbuminuria: marker of vascular dysfunction, risk factor for cardiovascular disease.
- **Monica Vermaa et al**[67]showed that marker of disease activity in rheumatic arthritis is microalbuminuria.

- **Hillege et al.**[68] conducted a cross-sectional cohort study in 2001 and reported the result. According to that microalbuminuria (defined as 20-200 mg/L) was detected in 7.2% (n=2,918) of study subjects and independently associated with increased cardiovascular morbidity.
- **Van de Wal et al.**[69] evaluated patients with chronic heart failure with age of 69 ± 12 years, and found that 32% of patients had microalbuminuria which is higher than in the general population;
- **Molitch, Market.al 2010 .**[70] showed that prevalence of Diabetes is high in patients with microalbuminuria and lower eGFR.
- **Sharan Badiger et.al** [71]**:** Microalbuminuria in essential hypertension increases the risk of developing target organ damage.

Detection of microalbuminuria

As excretion of albumin exhibits high variability due to many confounders the diagnosis of microalbuminuria should be ideally based on screening of multiple samples using either 24-hour urine collection or first-morning voids.

The various methods available for checking the presence and levels of microalbuminuria.

- **Dipstick method**

Sarafidis et al., 2007[73] showed that microalbuminuria can be detected by dipstick method

The various available options are there to detect microalbuminuria the advantage is that these strips are inexpensive and easy to use in clinical setting but may not provide precise quantitative measurement of albumin levels.

- **Immunoassay**

- **Chromatographic techniques**
  - **Size-exclusion high performance liquid chromatography**

- **Confounders of microalbuminuria**

Upright posture	Pregnancy
Exercise	Menstruation
Fever	Haematuria
Symptomatic UTI	Renal impairment
Heavy protein diet	Hyperglycemia
Inflammation	Hypertension
Infections (e.g. hepatitis)	Heart failure

Figure 7: Confounders of microalbuminuria [74]

- **Olivariuset al**[75]showed there is a direct correlation between body mass index and urinary albumin excretion

- **Poortmans**[76]in his study showed the relationship between physical exercise as it increases urinary albumin excretion in normal individuals
- There is an association of pregnancy and increase in urine albumin excretion [77]
- A reduction of dietary protein intake reduces microalbuminuria, as shown by **Cohen et al**[78]. Similarly in long-term studies, changes in dietary protein correlate with changes in urinary albumin excretion as shown by **Toeller, M., Buyken et.al**[79]
- **Mogensen**[72]showed that injection of dibasic amino acids acutely increases urinary albumin excretion.
- Stressful situations increase albumin excretion
- **Carter, Joanne L., et al**[80]stated that asymptomatic urinary tract infection increases albumin excretion.
- **Rosa, Tania Torres, and Paolo Palatini** [81]showed in essential hypertension urinary albumin excretion is directly proportional to the rise in BP values

Studies on ischemic stroke and association with microalbuminuria

In the **J Chowdhury**[82] people aged inbetween 45-70 years were included. 86% of MA +ve, and 63% MA –ve group were in 60 years of age group. 63.3% are males and 36% were females in MA +ve group. 13.3% are females and 86.7% are males in MA –ve group. The mean age and sex differences between MA +ve and MA –

ve patients were not significant. According to that study, mortality rate in MA +ve group was 26.7% and that of MA –ve group is 11.7%.

In **Ghosh et al**[83] study (prospective), acute ischemic stroke patients(who were not diabetic) were included, and two more groups were formed by 60 healthy individuals whose age, sex were matched with 70 patients who had chronic neurological disease. On day 1,4,7 they measured spot urinary albumin creatinine ratio in first morning sample. 61.79% of acute ischemic patients were MA +ve on day 1 when compared to 13% in non stroke neurological patients. It was only 7% in healthy controls. Patients with MA has 25-45% (high) 14 day disease specific mortality. Patients without it has only 5.88% mortality.

A study performed by **Klausen**[84], that gave results that MA is associated with increased mortality in patients with cardiovascular diseases. A timed overnight sample of urine were collected from 491 woman and men with coronary artery disease(age inbetween 30-80 years of age). 141 patients in that group died during the followup period. The death risk associated with MA were univariate cox proportional hazards regression analysis. 2.0 was relative risk($p<0.001$) of mortality in between the patient with UAE(Urinary Albumin Excretion) >5µg/min have high risk of death(100% high risk).

Gumbnger et al.[85]did a study to know potential of microalbuminuria as a prognostic marker in patient with acute ischemic stroke and he concluded that microalbuminuria is a strong predictor of poor outcome.

Beamer et al.[86] found that microalbuminuria is commonly found in patients with stroke and a main risk factor of stroke. Though other risk factors were corrected.

Lee et al.[87] concluded microalbuminuria independent and strong association with stroke risk. Meng Lee, Jeffrey L Saver et al, did a meta analysis in Los Angeles, they identified 12 studies, with total of 48,598 participants 1263 stroke events. And concluded presence of microalbuminuria was associated with higher risk of stroke after adjustment for cardiovascular risk factors are done.

Sander et al.[88] did a study on investigation of patients with ischemic stroke in neurological rehabilitation(INSIGHT).

A F Muhammedconducted a on patients of ischemic stroke, and found that micro albuminuria was present in 94(48.2%) patients and was absent in 101(51.8%) patients.[89]

In **M Heikki,** study he found out that there is significant association between proteinuria (different degrees) and incidence of stroke in both the non diabetic and diabetic patients provided adjustment for cardiovascular risk factors were done.[90]

Nancy Beamer, Bruce M Coull, Wayne M Clarke, Mike Wynn did a study in Portland in 1999, and found out that microalbuminuria was very much prevalent (3 times) in patients with stroke(mainly in recent stroke). The prevalence of microalbuminuria was high in all major ischemic stroke subtypes i,e atheroembolic,

dioembolic and lacunar. Microalbuminuria was an independent significant risk factor of future CVA provided all other risk factors were controlled [91].

Farooq et al did a cross sectional study from 1st Apirl 2009 -30th sep 2009 to determine the frequency of microalbuminuria in patient with ischemic stroke. Out of the 195 patients of ischemic stroke studied microalbuminuria was present in 94 and absent in 101.[92]

Dirk Sander et al[93], found that microalbuminuria has association with increase in thickness of intima and media of blood vessel wall, polyvascular disease, decrease in ankle brachial index.

Nidhinandana et al.[94] did a study during Oct 2007- Jan 2008. He determined the association (relationship) between risk factors for ischemic stroke in 173 patents older than 25 years and concluded that diabetic mellitus and hypertension are one of the important factor for ischemic stroke associated with microalbuminuria. And in this diabetes is the most important factor related to microalbuminuria in patients with ischemic stroke.

Das et al.[95] did a case control study from January 2008 to December 2010 about microalbuminuria as a risk for ischaemic stroke using urine spot analysis. 31.7% of the patient had microalbuminuria. But 8.3 % of the controls had that.

Burton et al.[96]**I**n 12 observational studies on 48,596 patients to know the relationship between microalbuminuria and overall stroke risk. Meta-analysis of

that studies found out that there is 92% increase in risk of stroke in patient with microalbuminuria provided adjustment for other cardiovascular risk factors was done.

MATERIALS AND METHODS

This study was a non-randomized cross sectional study done in medicine wards and IMCU of Sree Mookambika Institute of Medical Sciences, Kulasekharam during the 18 months study period on 50 ischemic stroke patients admitted and met pre-defined criteria gave informed consent for the study were chosen by convenient sampling technique. The study was initiated after obtaining ethical clearance from the institutions ethical clearance committee.

The Scientific basis of sample size used in the study was

$$\frac{4Pq}{d^2}$$

where p=prevalence q=100-P d=20% of P

Here P=69

$$\frac{4*69(100-69)}{(69*20\ 100)^2} = \frac{8556}{190.44} = 45$$

Even if the sample size is 45, for convinence 50 patients were studied.

The criteria were

a) Inclusion Criteria:

- Patients with history of acute stroke presenting within 24 hours, confirmed by computer tomography of brain in SMIMS Kanyakumari, was included in the study.
- Age more than 45 years

- Both Sex (Male & Female)

b) Exclusion Criteria:

- Patients who were not willing
- Kidney diseases both acquired and congenital
- Liver disease
- Chronic inflammatory gastrointestinal disorder
- Neoplasms
- Those on NSAID's or other immunosuppressant's
- Coronary artery disease or acute coronary event.

After acceptation of the thesis by the ethics committee,

An informed consent was obtained from all ischemic stroke patients who came to the IMCU and Medicine wards.

A detailed general physical examination, Vitals and a detailed CNS examination was performed.

Complete blood picture, Serum electrolytes, Blood urea, Random plasma glucose, FBS PPBS in case of diabetics, Serum creatinine, Fasting lipid profile, Electrocardiogram, Chest X-ray, CT scan brain- plain (contrast if needed)MRI brain (if indicated)2D echo (if indicated)Colour Doppler (if indicated) were obtained from case sheet.

- Then the urine albumin level of each patient was estimated and patients who were having urine albumin in microalbuminuric range and not having microalbuminuria are separated.
- Then the strength of association of Microalbumunria in incidence of acute ischemic stroke was calculated and its association with risk factors estimated.

Another objective of my study was to find relationship between microalbuminuria and subtype of stroke. For this, the CT finding of whether it is a Middle cerebral artery or Anterior cerebral artery or Posterior cerebral artery involvement was found out and its correlation with microalbuminuria was studied.

- **Sample collection and storage:**

Urine samples was collected in autoclaved, dry, capped glass bottles. At least 10 ml of random, midstream urine samples were collected from patients and centrifuged albumin as centrifuged albumin can be stored 7 days @ 2-8^{o}c

- **Laboratory investigations:**

A spot urine sample collected was centrifuged prior to testing.

The Reagents A and B were used for assessment of urinary microalbumin levels kept at 2-8°C. Urine samples were subjected to analysis by the Latex Method for quantitative assessment of the amount of microalbumin (mg/L). This was done by a semi-automated biochemistry analyzer (Star 21 Plus).

Depending on urine albumin excretion, microalbuminuria (30-299µg/mg creatinine) was identified.

All the investigations were carried out in the Central Laboratory, Sree Mookambika Institute of Medical Sciences, Kulasekharam

DATA AND STATISTICAL ANALYSIS

All the data collected were in Microsoft Excel spread sheet.

The data was expressed in MEAN±SD. Statistical Package for Social Sciences (SPSS 16.0) version used for analysis. Chi-square applied to find the statistical significant between the groups. P value less than 0.05 considered statistically significant at 95% confidence interval.

Statistical Analysis was done using SPSS software version 23.0. In that statistical analysis, p value <0.05 is considered significant.

ANALYSIS AND INTERPRETATIONS

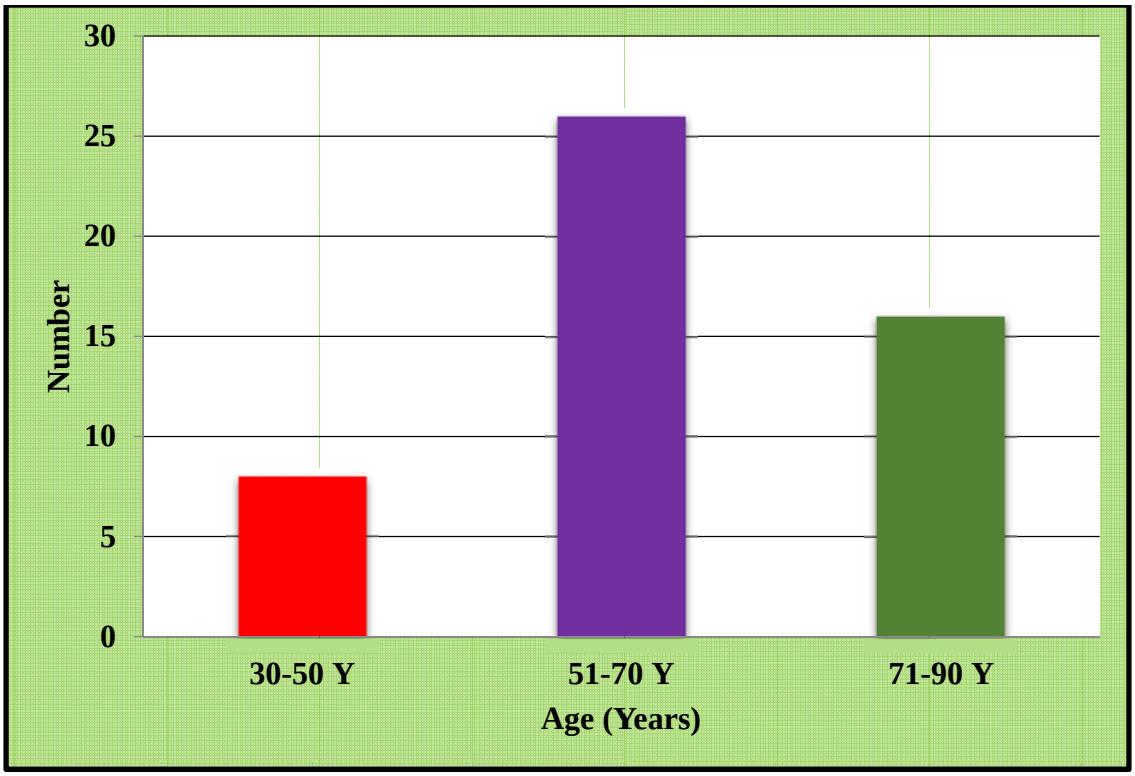
Non-randomized cross-sectional study done in the medicine wards and IMCU of Sree Mookambika Institute of Medical Sciences, Kulasekharam during the 18 months study period on 50 ischemic stroke patients admitted and met pre-defined criteria. These were our observations

DEMOGRAPHIC DATA

AGE DISTRIBUTION

AGE	Frequency	Percent
<44	2	4
45-50	6	12
51-55	6	12
56-60	8	16
61-65	5	10
66-70	7	14
71-75	7	14
>75	9	18
total	50	100

Table 2: AGE DISTRIBUTION



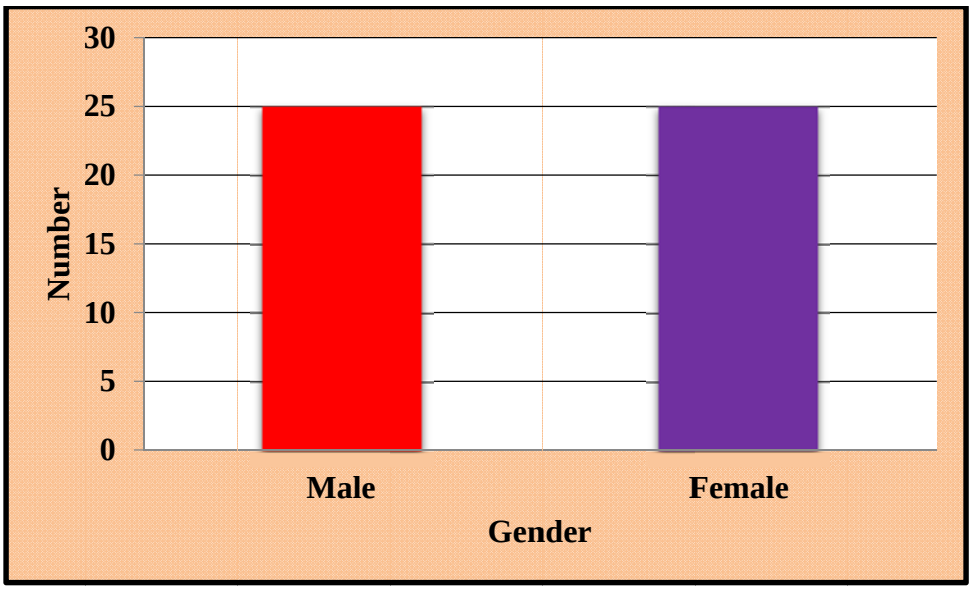
Graph-1: Distribution of patients based on the age

In our study most of the patients come under the age group of 51 to 70 years (52%).

GENDER DISTRIBUTION

	Frequency	%	Valid%	Cumulatve %
Female	25	50	50	50
Male	25	50	50	100.0
Total	50	100.0	100.0	

Table 3: **GENDER** DISTRIBUTION



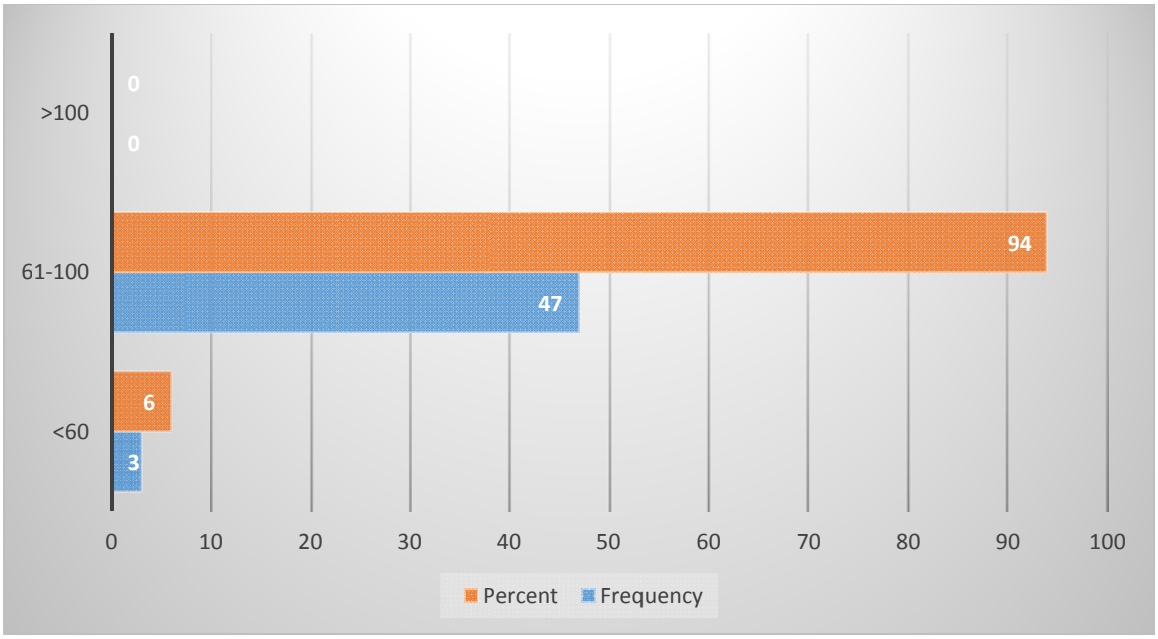
Graph-2: Distribution of patients based on gender

There were equal number of males and females in the study

Signs

Pulse (bpm)	Frequency	Percent
<60	3	6
61-100	47	94
>100	0	0

Table 4: Pulse



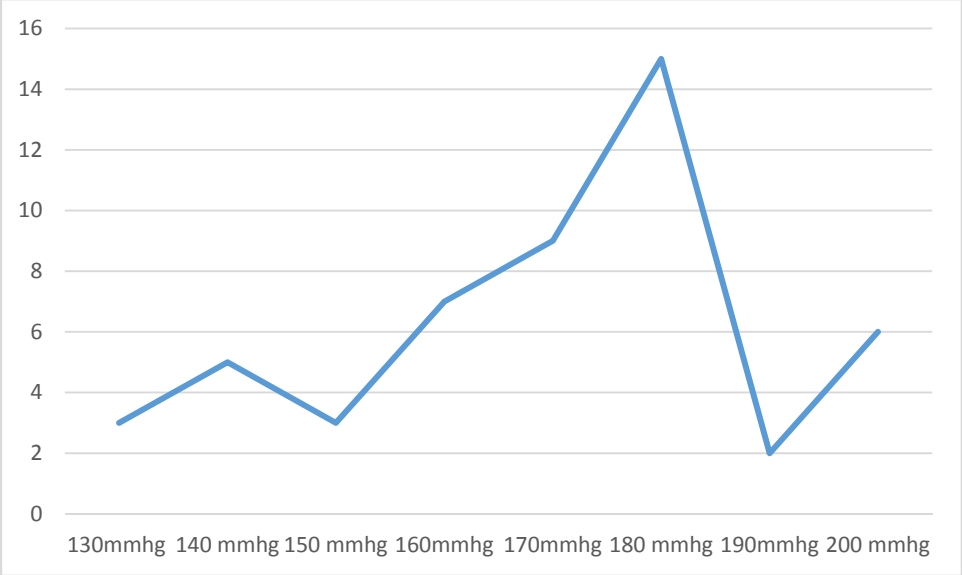
Graph 3: **Pulse**

There was no much abnormality of the pulse rate in our study 94% were in the normal range and 6% had bradycardia

Systolic BP

Valid	frequency	%	Valid %	Cumulative %
130	3	6.0	6.0	6.0
140	5	10.0	10.0	16.0
150	3	6.0	6.0	22.0
160	7	14.0	14.0	36.0
170	9	18.0	18.0	54.0
180	15	30.0	30.0	84.0
190	2	4.0	4.0	88.0
200	6	12.0	12.0	100.0
Total	50	100.0	100.0	

Table 5: **Systolic BP**



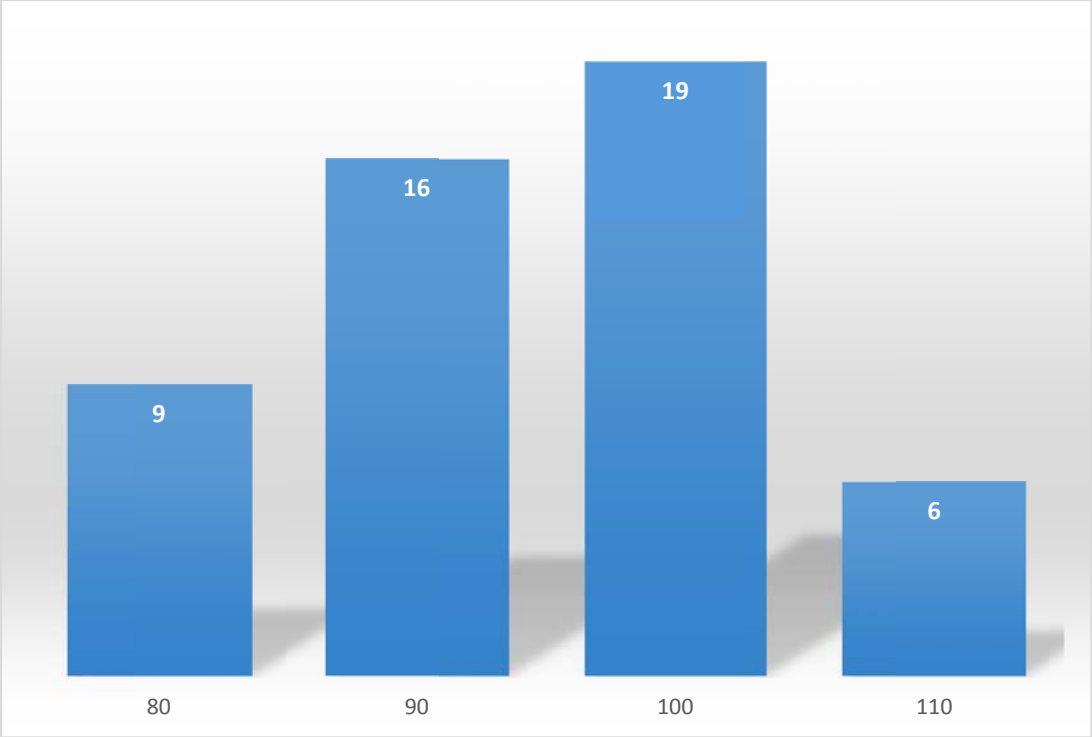
Graph 4: **Systolic BP**

Most of the patients were having Systolic blood pressure between 180-190 mmHg.

Diastolic BP

Valid	frequency	%	Valid %	Cumulative %
80	9	18.0	18.0	18.0
90	16	32.0	32.0	50.0
100	19	38.0	38.0	88.0
110	6	12.0	12.0	100.0
Total	50	100.0	100.0	

Table 6: Diastolic BP



Graph 5: Diastolic BP

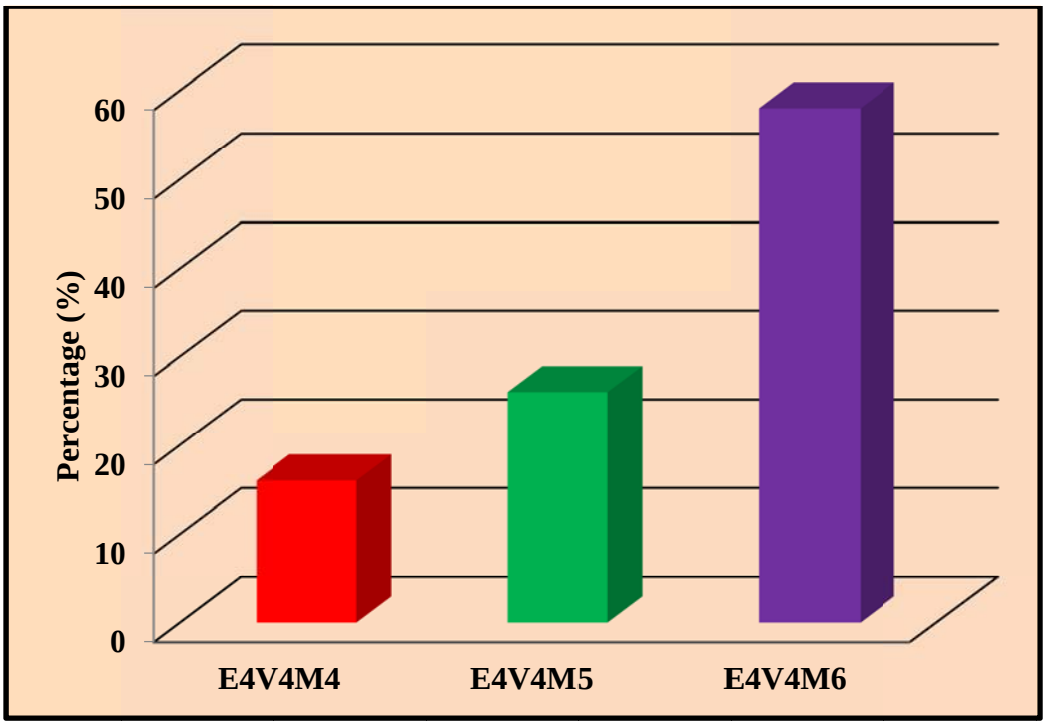
80% of the patients had elevated diastolic blood pressure.

GCS

GCS	Number	Percentage (%)
E4V4M4	8	16.00
E4V4M5	13*	26.00
E4V4M6	29*	58.00

(*p<0.05 significant)

Table 7: Distribution of patients based on GCS

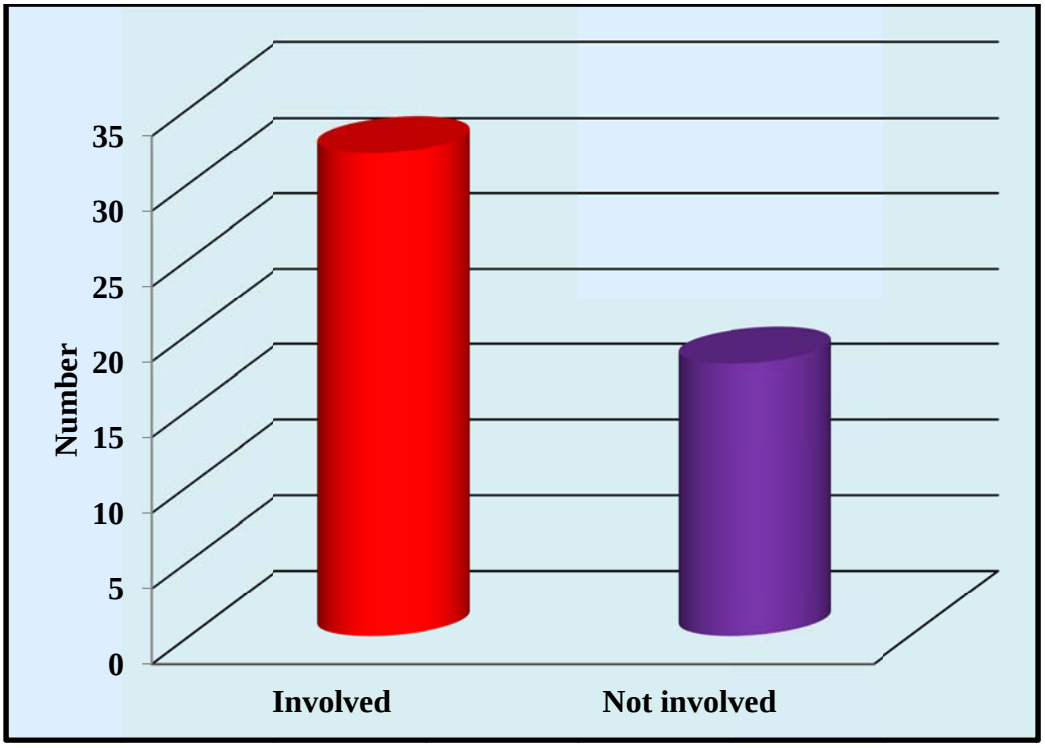


Graph 6: Distribution of patients based on GCS

CRANIAL NERVE INVOLVEMENT

Cranial Nerve involvement	Number	Percentage (%)
Involved	32	64.00
Not involved	18	36.00

Table 8: Distribution of patients based on Cranial Nerve involvement



Graph 7: Distribution of patients based on Cranial Nerve involvement

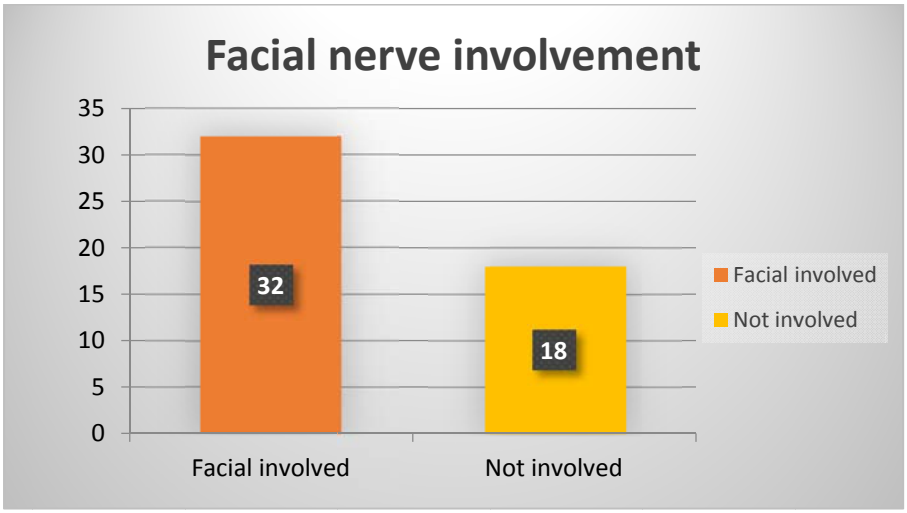
64% of the patients had cranial nerve involvement.

Facial nerve involvement

Nerve involved	Number	Percentage (%)
Facial nerve	32	64.00
Not facial nerve	18*	36.00

(*p<0.05 significant compared to facial with not facial nerve)

Table 9: Distribution of patients based on nerve involved



Graph 8: Involvement of facial nerve

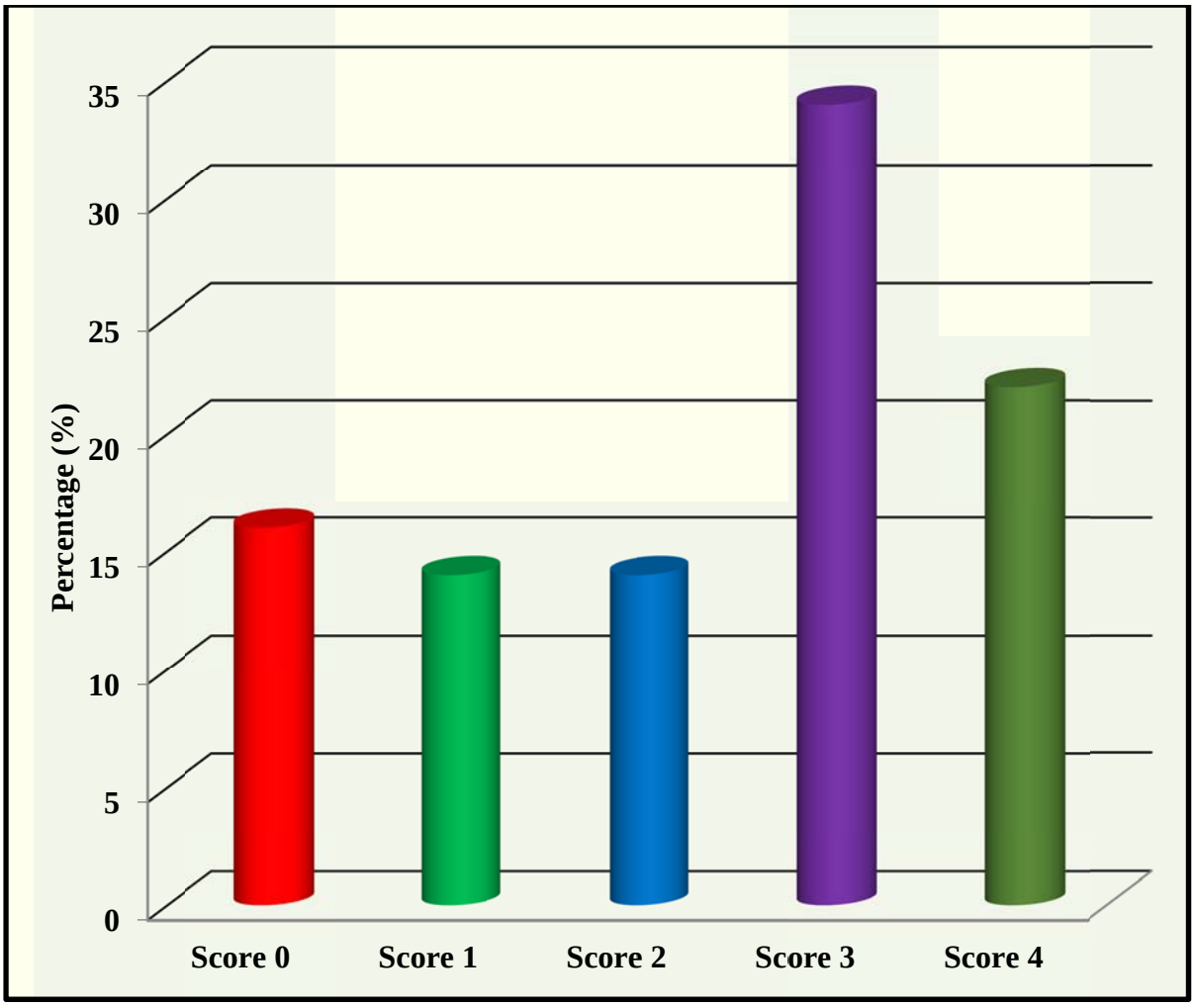
Facial nerve involvement was seen in 64% of all cases studied. There was statistically significant involvement of facial nerve among other cranial nerves in acute ischaemic stroke.

Motor grade in affected limb

Motor system power of affected limb	Number	Percentage (%)
Grade 0	8	16.00
Grade 1	7	14.00
Grade 2	7	14.0
Grade 3	17*	34.00
Grade 4	11	22.00

(*p<0.05 significant compared score 3 with others)

Table 10: Distribution of patients based on motor system power of affected limb



Graph 9: Distribution of patients based on motor system power of affected limb

Most of the patients had a power of Grade III(34%). 16% of the patients had Grade 0 power.

BIOCHEMICAL INVESTIGATION

Biochemical investigation	MEAN±SD
RBS (mg/dl)	154.72±5.25
Urea (mg/dl)	24.12±8.44
Creatinine (mg/dl)	0.90±0.22
Total cholesterol (mg/dl)	176.62±1.77
Triglycerides (mg/dl)	128.12±1.92
LDL	138.90±9.76
HDL	63.36±9.51

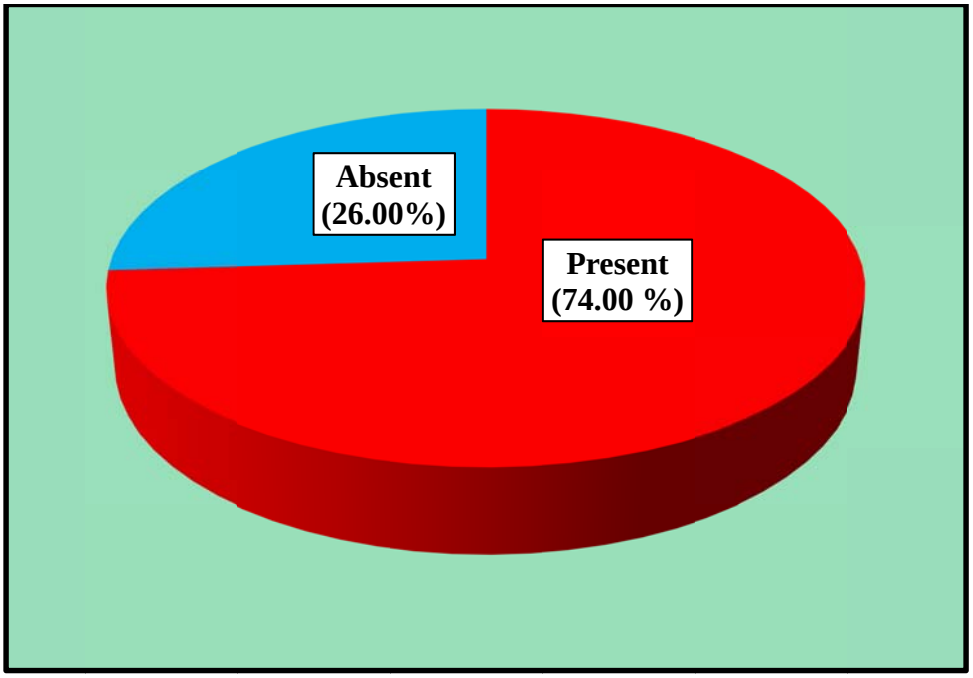
Table 11: Mean values of biochemical investigation

URINE MICROALBUMIN

Urine microalbumin	Number	Percentage (%)
Present	37	74.00
Absent	13*	26.00

(*p<0.05 significant compared present with absent)

Table 12: Distribution of patients based on presence of urine microalbumin



Graph 10: Distribution of patients based on presence of urine microalbumin

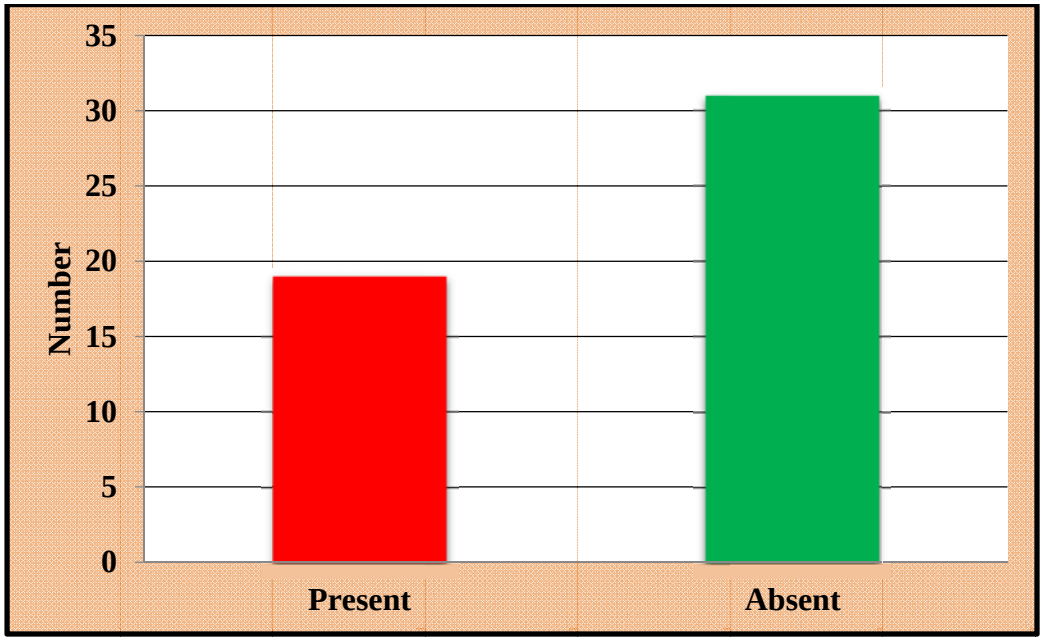
In our study, 74% of the patients had microalbuminuria and there was a statistically significant correlation between acute ischaemic stroke and urine microalbumin.

ECG showing LVH

ECG(LVH)	Number	Percentage (%)
Present	19	38.00
Absent	31*	62.00

(*p<0.05 significant compared present with absent)

Table 13: Distribution of patients based on ECG (LVH) changes



Graph 11: Distribution of patients based on ECG (LVH) changes

38% of the patients had LVH. There was higher incidence of microalbuminuria in patients with LVH which was statistically proven.

TERRITORY OF BRAIN INFARCT

Territory	Number	Percentage (%)
MCA	32	64.00
PCA	10	20.00
ACA	8	16.00

Table 14: Distribution of patients based on territory

Of the 50 patients with acute ischaemic stroke 32 had MCA infarct which accounts for 64%.

Microalbunuria	MCA		ACA		PCA	
	Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
Present	20	62.50	7	87.50	10	100.00
Absent	12*	37.50	1*	12.50	0*	00.00
Total	32	100.00	8	100.00	10	100.00

(*p<0.05 significant compared Present with Absent)

Table 15: Distribution of patients based on microalbuminuria in relation to territory

AGE DISTRIBUTION

Microalbuminuria	30-50 years		51-70 years		71-90 years	
	Number	**Percentage (%)**	**Number**	**Percentage (%)**	**Number**	**Percentage (%)**
Present	6	75.00	18	69.23	13	81.25
Absent	2	25.00	8	30.77	3	18.75
Total	8	100.00	26	100.00	16	100.00

Table 16: Distribution of patients based on microalbuminuria in relation to age

The mean age of the patients were 51 to 70 years. There was no significant correlation microalbuminuria with particular age group.

GENDER DISTRIBUTION

Microalbuminnuria	Male		Female	
	Number	Percentage (%)	Number	Percentage (%)
Present	18	72.00	19	76.00
Absent	7	28.00	6	24.00
Total	25	100.00	25	100.00

Table 17: Distribution of patients based on microalbuminuria in relation to gender

In our study, 25 males and 25 females were taken. There was no significant correlation between microalbuminuria and gender.

PULSE RATE AND BLOOD PRESSURE

Microalbunuria	**Pulse rate (MEAN±SD)**	**SBP (MEAN±SD)**	**DBP (MEAN±SD)**
Present	74.18±1.08	175.95±1.46*	97.02±8.11*
Absent	77.23±4.86	150.77±2.06	86.92±8.54

(*p<0.05 significant compared Present with Absent)

Table 18: Blood pressure in patients with and without microalbuminuria

The average systolic blood pressure of the patients with microalbuminuria was 175.95±1.46. There was significant correlation of microalbuminuria with elevated blood pressure.

BLOOD SUGAR

Microalbunuria	RBS (MEAN±SD)
Present	151.95±4.84*
Absent	162.62±6.43

(*p<0.05 significant compared Present with Absent)

Table 19: Blood sugar in patients with and without microalbuminuria

There was significant association of microalbuminuria with elevated blood sugar levels.

LIPID PROFILE

Microalbunuria	Total Cholesterol (MEAN±SD)	Triglycerides (MEAN±SD)	LDL (MEAN±SD)	HDL (MEAN±SD)
Present	176.03±1.59*	127.16±2.05*	137.73±1.07*	61.70±8.39
Absent	178.31±2.28	130.85±1.54	140.69±7.56	69.07±1.06

(*p<0.05 significant compared Present with Absent)

Table 20: Lipid profile in patients with and without microalbuminuria

RENAL FUNCTION

Microalbunuria	Creatinine (MEAN±SD)
Present	0.92±0.21*
Absent	0.86±0.23

(*p<0.05 significant compared Present with Absent)

Table 21: Creatinine levels in patients with and without microalbuminuria

DISCUSSION

With the increasing life expectancy and increase in the modifiable and non-modifiable risk factors, there has been an upsurge in the non-communicable diseases among them stroke is considered as diseases that has to be identified early or if possible be prevented so that the disability related it is reduced. Micro albuminuria is considered as a manifestation of a generalized disorder and its role in early prediction of various studies has been well documented .we conducted a study titled “A STUDY ON MICROALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS” which was done as a non-randomized cross sectional study in the medicine wards and IMCU of Sree Mookambika Institute of Medical Sciences, Kulasekharam for 18 months study period on 50 ischemic stroke patients admitted and met pre-defined criteria gave informed consent after obtaining ethical clearance from the institutions ethical clearance committee.

Urine microalbumin	Systolic BP	Diastolic BP	GCS	CN. Involved/ not	which nerve	Motor system	RBS(mg/dl	Creatinine	TC/ TG
Pearson Correlation	.624**	.420**	.092	.350*	-.350*	-.260	.181	.106	-.110
Sig. (2-tailed)	.000	.002	.524	.013	.013	.069	.208	.462	.448
N	50	50	50	50	50	50	50	50	50

Table 22: correlations of microalbuminuria with various parameters

Correlation of microalbuminuria with age,

The mean age in our study most of the patients were in the age of 51 to 70 years

There was no statistically significant correlation of microalbuminuria with age.

It is in correlation with the study done by Jarrett.et al [97]and Dogra et.al [98]who found no statistically significant correlation of microalbuminuria with age

In our study we found as the age advances the chances of stroke increase as in the study by Subramanyam [99]who found statistically significant **correlation** between increasing age in ischemic stroke

Correlation of microalbuminuria with gender,

There was a no correlation of microalbuminuria with gender.

It is in correlation with the study done by Chowta et.al [36]who found no statistically significant correlation of microalbuminuria with gender.

In our study we found that the both male and female gender has equal risk for chances of stroke. It is comparable with the study done by Subramanyam[99] who found statistically significant **correlation** of gender with ischemic stroke.

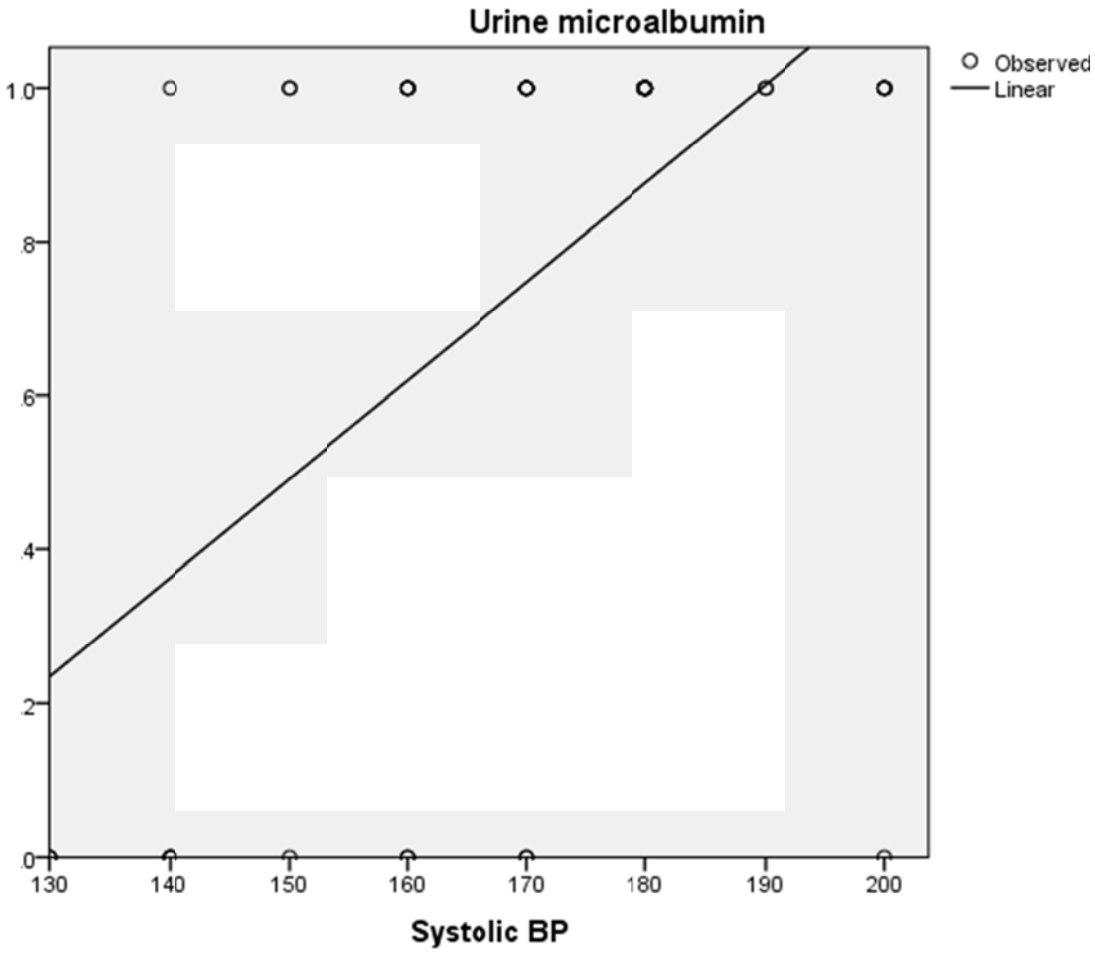
Total no. of patients in study: 50

No. of male patients : 25

No. of female patients : 25

Correlation of microalbuminuria with blood pressure

There was a positive correlation of microalbuminuria with Systolic BP and Diastolic BP. In our study of 50 patients with acute ischaemic stroke, most of the patients had a systolic blood pressure of 180-190 mmHg and diastolic blood pressure between 90-110 mmHg. And there was statistically significant correlations which were 0.624 and 0.420 with a p value<0.001.



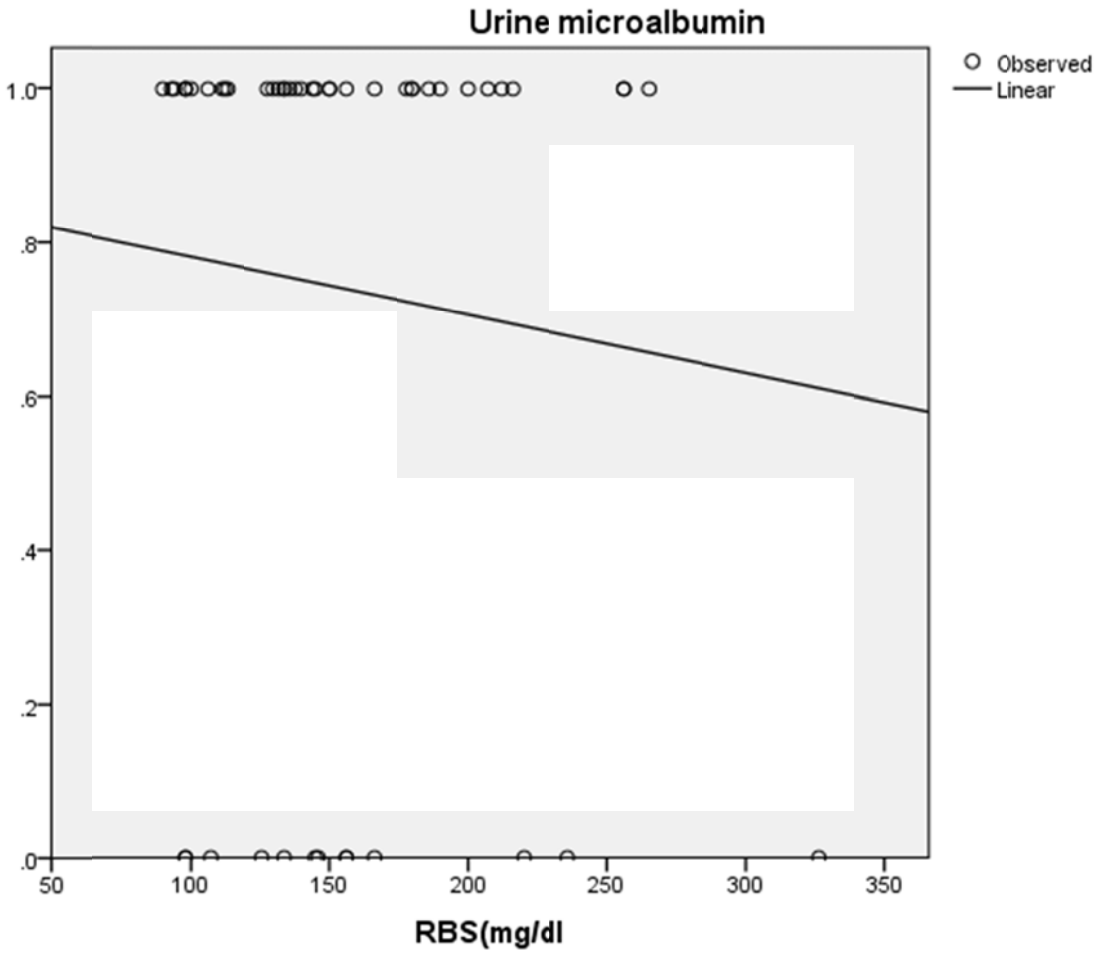
Graph 12: Correlation of microalbuminuria with blood pressure

- **Rosa, Tania Torres, and Paolo Palatini** [81]showed in essential hypertension urinary albumin excretion is directly proportional to the rise in BP values
- In the **Danish MONICA**[100] project showed that among the hypertensive subjects who developed future ischaemic disease the prevalence of microalbuminuria was higher.
- **Sharan Badiger et.al** [101]**:** Microalbuminuria in essential hypertension increases the risk of developing target organ damage

Correlation of microalbuminuria with RBS,

There positive correlation of microalbuminuria with the level of sugars in 37 patients who had an evidence of albumin in urine 10 had compared glucose tolerance and 26 had diabetes mellitus.

In our study, the total number of acute ischaemic stroke patients was 50 and the mean RBS value was 151.95±4.84 and there was significant correlation between microalbuminuria and blood sugar level.



Graph 13: Correlation of microalbuminuria with RBS

The correlation of microalbuminuria with diabetes mellitus was studied in various studies mentioned below

- A study **Diercks et al**[102]of 64 asymptomatic Diabetes Mellitus (type I patients) had showed a greater incidence of myocardial ischemia, which was detected by electrocardiography and stress echocardiography. In that patients, incidence of myocardial ischemia is more in the presence of microalbuminuria(25%) as compared to the patients with normoalbuminuria(6.3%)(OR 6.3; 95% CI 1.2 to 37.8; P = 0.03).

- **Molitch, Market.al 2010** [103] showed that microalbuminuria +ve patients have high prevalence of diabetes & lower eGFR value.

  Correlation of microalbuminuria with ECg finidings

- **Berton et al**[96]showed the importance of microalbuminuria as prognostic factor for short and long-term outcomes of acute myocardial infarction
- **Koulouris et al., 2005**[104] and **Apostolovic et al., 2011**[105]showed that increased urinary excretion of albumin was a strong predictor for myocardial infarction complications including mortality.
- ECG images from 7579 PREVEND participants who were not diabetic revealed an independent relationship between microalbuminuria and infarct patterns (odds ratio [OR] 1.61; 95% CI 1.12 to 2.32), major ischemia (OR 1.43; 95% CI 1.08 to 1.91), and minor ischemia (OR 1.32; 95% CI 1.03 to 1.68[106]

Correlation of microalbuminuria with lipid profile

In our study, no. of acute ischaemic stroke patients: 50

Mean value for total cholesterol : 176.03±1.59

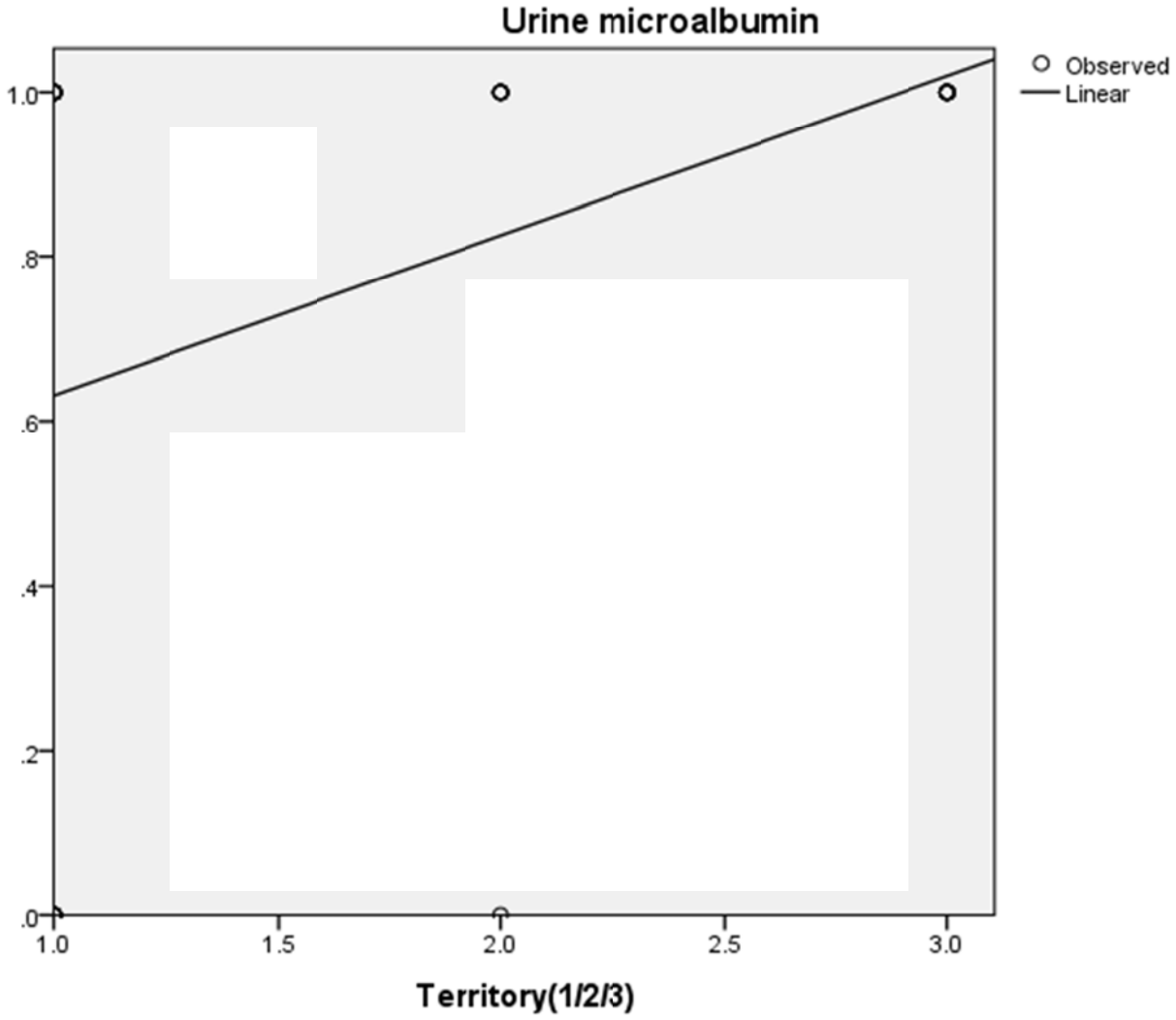
Mean value for triglycerides : 127.16±2.05

Mean value for LDL : 137.73±1.07

There was statistically significant correlation between microalbuminuria and lipid profile.

Chowdhury et al in their study found a positive Correlation between microalbuminuria & lipid profile in acute ischemic stroke which is comparable to our study [82]

Correlation of microalbuminuria with vascular territory,



Graph 14: Correlation of microalbuminuria with vascular territory

Coefficients

	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	T	Sig.
Territory (1-MCA/2-ACA/3-PCA)	.194	.074	.356	2.641	.011
(Constant)	.437	.129		3.383	.001

In our study of 50 patients with acute ischaemic stroke, microalbuminuria was found equally in all three vascular territories(MCA, ACA and PCA). Thus, there was no significant correlation of microalbuminuria with particular vascular territory.

RESULTS

In our study titled ‘A STUDY ON MICRO ALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS’ 50 cases of acute ischemic stroke patients were taken.

Among the 50 patients, most of the patients come under the age of 51 to 70 years(52%). There was no statistically significant correlation of microalbuminuria with age.

There were 25 male and 25 female participated in the study. We found that the both male and female gender had equal risk for chances of stroke.

In our study, we found that the prevalence of microalbuminuria in patient with acute ischemic stroke was 74% and there was significant correlation between microalbuminuria and stroke. Thus we concluded that there was a strong possible relation of microalbuminuria as a risk factor for stroke.

The cranial nerve involvement was seen in about 32 patients(64%) of all the 50 patients. There was a significant correlation of facial nerve involvement(32 cases-64%) with acute ischaemic stroke than other cranial nerves.

The most common vascular territory involved was MCA about 64%. In our study of 50 patients with acute ischaemic stroke, microalbuminuria was found equally in all three vascular territories(MCA, ACA and PCA). Thus, there was no significant correlation of microalbuminuria with particular vascular territory.

38% of the patients had LVH. There was higher incidence of microalbuminuria in patients with LVH which was statistically proven.

In our study of 50 patients with acute ischemic stroke, most of the patients had a systolic blood pressure of 180-190 mmHg and diastolic blood pressure between 90-110 mmHg. There was a positive correlation of microalbuminuria with Systolic BP and Diastolic BP.

There was statistically significant correlation between microalbuminuria and lipid profile.

There was significant correlation between microalbuminuria and blood sugar level.

CONCLUSION

In our study titled “A STUDY ON MICROALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS” we found that

- the prevalence of microalbuminuria in patients with acute ischemic stroke was 74%. there was a strong positive relationship of microalbuminuria as an important independent risk factor for stroke. Significant correlation between various risk factors of ischemic stroke such as hyperglycemia, hypertension, hyperlipidemia with microalbuminuria has established. Eventhough microalbuminuria was present in 74% of my study, no significant association of microalbuminuria with particular vascular territory has noted. But we recommend that larger trials and long term follow up studies be conducted

DRAWBACKS:

- Small group taken into study.
- Long term follow-up have not conducted.
- Only spot examination of the blood sugar and blood pressure are noted.
- Though hyperglycemia and hypertension are one of the causes of microalbuminuria, patients with these diseases are not excluded in this study.

SUMMARY

We conducted a study titled “A STUDY ON MICROALBUMINURIA IN ACUTE ISCHEMIC STROKE PATIENTS” which was done as a non-randomized cross sectional study in the medicine wards and IMCU of Sree Mookambika Institute of Medical Sciences, Kulasekharam for 18 months study period on 50 ischemic stroke patients admitted and met pre-defined criteria gave informed consent after obtaining ethical clearance from the institutions ethical clearance committee.

In our study of 50 patients with ischaemic stroke, 74% had significant microalbuminuria. MCA was the most common territory involved with 64% cases followed by PCA in 10 % and ACA IN 8%. But no significant correlation of microalbuminuria with particular vascular territory has found out. In our study 62 % cases had ECG showing LVH pattern. crainal nerve involvement most commonly facial nerve was seen in 64% of all cases. There was a positive co-relation between lipid profile and LVH with microalbuminuria . There is statistically significant co-relation between elevated blood sugar and elevated blood pressure in patients admitted with ischemic stroke less than 24hours. There was strong positive relationship of microalbuminuria in acute ischemic stroke patients and microalbuminuria is an important independent risk factor for stroke.

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APPENDICES

ETHICAL CLEARANCE CERTIFICATE

Sree Mookambika Institute of Medical Sciences
Kulasekharam (K.K District, TN) 629161

Phone No: 04651-280866, Fax No. 04651-280740

Institutional Human Ethics Committee

Registered under CDSCO with Reg No. ECR/446/Inst/TN/2013

Ref. No. SMIMS/IHEC/2015/A/06 **Date: 10th April 2015**

Certificate

This is to certify that the Research Protocol Ref. No. **SMIMS/IHEC/2015/A/06**, entitled "A Study on Microalbuminuria in Acute Ischemic Stroke Patients" submitted by Dr. Jayaram J. K, Postgraduate of Department of General Medicine, SMIMS has been approved by the Institutional Human Ethics Committee at its meeting held on 13th of March 2015.

[This Institutional Human Ethics Committee is organized and operates according to the requirements of ICH-GCP/GLP guidelines and requirements of the Amended Schedule-Y of Drugs and Cosmetics Act, 1940 and Rules 1945 of Government of India.]

IHEC Date 13/04/2015

Dr. Rema Menon. N
Member Secretary
Institutional Human Ethics Committee
Professor of Pharmacology and HOD
SMIMS, Kulasekharam [K.K District]
Tamil Nadu -629161

18. INVESTIGATOR DECLARATION

- I certify that the research proposal here is not necessarily the duplicate of previously reported research
- I will obtain approval from Institution Research Committee (IRC) & Institution Human Ethics Committee (IHEC) of SMIMS, before starting my research proposal.
- I will obtain the approval from IRC and IHEC before initiating any significant changes in the study
- I certify that performances of research will be carried out in accordance with the GCP & GLP guidelines laid by international & national organizations from time to time.
- I will maintain all records pertaining to my research activities and will produce before the IHEC for scrutiny if required.

Date:

Place:

Signature of Principal Investigator

Dr Jayaram J.K.

Post Graduate, Department of General Medicine

Sree Mookambika Institute of Medical Sciences

Kulasekharam, 629161

Mobile Number: 09566563322

e-mail:jayaramj24@gmail.com

Signature of Guide: Dr Thilagar

Professor , Department of General Medicine

Sree Mookambika Institute of

Medical Sciences

Kulasekharam, 629161

Contact No:9367524551

Email id:dr.thilagar55@gmail.com

Signature of HOD

Dr.Kaniraj Peter MD

CONSENT FORM

PART 1 OF 2

INFORMATION FOR PARTICIPANTS OF THE STUDY

Dear Volunteers,

We welcome you and thank you for your keen interest in participating in this research project. Before you participate in this study, it is important for you to understand why this research is being carried out. This form will provide you all the relevant details of this research. It will explain the nature, the purpose, the benefits, the risks, the discomfort, the precautions and the information about how this project will be carried out. It is important that you can read and understand the contents of the form carefully. This form may contain certain scientific terms and hence, if you have any doubts or if you want more information, you are to ask the study personnel or the contact person mentioned below before you give your consent and also at any time during the entire course of the project.

1. **Name of the Principal Investigator:** Dr.Jayaram J k

   Postgraduate – M.D General Medicine

   SreeMookambika Institute of Medical Sciences

   Kulasekharam

2. **Name of the Guide:** Dr.Thilagar

Professor

Department of General Medicine

SreeMookambika Institute of Medical Sciences

Kulasekharam

3. **Institute: details with Address:** SreeMookambika Institute of Medical Sciences Kulasekharam

Kanyakumari District-629161

Tamil Nadu

4. **Title of the study: STUDY ON MICROALBUMINURIA IN ISCHEMIC STROKE PATIENTS AT SMIMS KULASEKHARAM**

5. **Background Information:**

Microalbuminuria, a marker of endothelial dysfunction is associated with global vascular risk but the nature and magnitude of link between microalbuminura and incident stroke has not been clearly defined.

Purpose of the study is to assess the strength of association between microalbuminuria and incidence of ischemic stroke.

6. Aims and Objectives:

To study incidence of microalbuminuria in patients with acute ischemic stroke that is less than 24 hours. (small vessel or large vessel disease)

Relationship of microalbuminuria to risk factor for stroke.

Prevalence of microalbuminuria in major subtypes of ischemic strokes.(Small vessel or large vessel disease).

7. Scientific justification of the study:

Assessing Microalbinuria using simple test could be a window to systemic vasculature in general and individuals susceptible to target organ damage. There is convincing evidence of an independent positive relationship between overt proteinuria and stroke risk,but the nature and magnitude of the link between microalbuminuria and incident stroke has so far not been systematically investigated. In this study,we aim to assess the consistency and strength of the association of microalbuminuria with incidence of ischemic stroke and association with risk factors.

8. Procedure of the study: Patients with acute ischemic stroke confirmed by CT of the brain admitted to medical ward of SMIMS Kulasekharam will be are includedA detailedmedical history willbe taken and a detailed general physical examination will be performed.Vitals recording and detailed CNS examination and examination of other systems will be done.Urine samples to assess urinary albumin level will be sent to central lab – where urine spot examination in semi-automatic analyser will be done to assess urine albumin level.

9. **Expected risk of the participants:**No risk

10. **Expected Benefits of the Research for the participants:**Screening for microalbuminuria.

11. **Maintenance of confidentiality:**

All data collected for the study will be kept confidential and will only reflect on the general statistical evaluation but will not reveal any personal details.

12. **Why have I been chosen to be in this study:**

You have ischemic stroke and fulfill the criteria of selection

13. **How many people will be in the study:**50

14. **Agreement of compensation to the participants:**No

15. **Anticipated prorated payment, if any,to the participants of the study:**Nil

16. **Can I withdraw from study at any time during the study period:**Yes

17. **If there is any new finding/information, would I be informed:**Yes

18. **Expected duration of the participants participation in the study:**Single visit

19. **Any other pertinent information:**No

20. **Whom do I contact for further information:**Dr. Jayaram JK

For any study related queries,you are free to contact:

Dr. JAYARAM J K

Post Graduate – DEPARTMENT OF GENERAL MEDICINE

SreeMookambika Institute of Medical Sciences,

Kulasekharam

Mobile number: 09566563322

e-mail: jayaramj24@gmail.com

Place:

Date:

Signature of Principal Investigator

Signature of Participant

CONSENT FORM

PART 2 OF 2

PARTICIPANTS CONSENT FORM

The details of the study have been explained to me in writing and details have been fully explained to me. I am aware that the results of the study may not be directly beneficial to me but will help in the advancement of medical sciences. I confirm that I have understood the study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reasons, without the medical care that normally is provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have given details of the study. I fully consent to participate in the study titled Microalbuminuria in acute Ischemic stroke in a tertiary medical care centre, Kulasekharam.

Serial no/Reference no:

Name and Address of the participant:

Contact number of the Participant:

Signature/Thumb impression of the participant/Legal guardian

Witness

1.
2.

Date:

Place:

List of graphs

Figures

Tables

S.No.	Name	IP No.	Age/sex	Presenting Complaints	Pulse(bpm)	BP mmHg	GCS	HMF conscious	CN involvement	Which nerve	Motor system	Sensory system	Meningeal involvement	Skull & spinal cord	CVS (S1S2)	RS (B/l NVBS)	Abdomen	RBS(mg/dl)	Urine microalbumin	Urea (mg/dl)	Creatinine (mg/dl)	TC/TG	LDL/HDL	ECG(LVH)	CTbrain infarct	Territory
1.	Saraswathy	1616698	57 /F	W	78	160/100	E4V4M6	C	I	F	3	NA	A	N	+	+	N	146	A	17	0.6	203 /14	141/ 80	A	P	M
2.	Sasthra Kani	1614943	50 /F	W	82	180/110	E4V4M5	C	I	F	1	NA	A	N	+	+	N	112	P	21	0.8	188 /13	150/ 62	P	P	M
3.	Palammal	1610701	78 /F	W	76	140/80	E4V4M6	C	I	F	3	NA	A	N	+	+	N	126	A	16	0.7	150 /13	130/ 56	A	P	M
4.	Chellamma	1612176	80 /F	W	90	160/90	E4V4M4	C	NI		3	NA	A	N	+	+	N	186	P	11	1	170 /98	140/ 69	A	P	P
5.	Appu	1612710	68 /M	W	84	130/80	E4V4M6	C	I	F	4	NA	A	N	+	+	N	98	A	09	0.8	200 /14	149/ 88	A	P	M
6.	Selva Raj	1614221	46 /M	W	68	170/100	E4V4M6	C	I	F	0	NA	A	N	+	+	N	216	P	30	1	164 /10	143/ 68	A	P	M
7.	Ravi	1614141	52 /M	W	78	160/100	E4V4M6	C	I	F	4	NA	A	N	+	+	N	166	P	23	1.2	159 /13	137/ 72	A	P	M
8.	Vijayakumar	1613646	79 /M	W	80	180/110	E4V4M5	C	NI		2	NA	A	N	+	+	N	145	P	39	0.8	176 /10	142/ 80	P	P	A
9.	Sulochana	1617110	80 /F	W	94	170/100	E4V4M4	C	NI		4	NA	A	N	+	+	N	190	P	15	0.8	184 /12	127/ 64	P	P	P
10	Senthil Murugan	1613829	46 /M	W	72	200/100	E4V4M5	C	I	F	3	NA	A	N	+	+	N	220	A	17	0.6	192 /13	138/ 64	P	P	M
11	Sreekumari	1617220	49/ F	W	74	170/90	E4V4M6	C	I	F	4	NA	A	N	+	+	N	145	A	21	1.2	202 /14	141/ 55	P	P	M
12	Manikandan	15213875	46/ M	W	78	160/100	E4V4M6	C	I	F	3	NA	A	N	+	+	N	256	P	24	1.3	190 /13	132/ 64	A	P	M
13	Chandra	15213977	39/ F	W	68	180/100	E4V4M5	C	NI		3	NA	A	N	+	+	N	98	P	22	1	185 /12	112/ 58	A	P	A
14	Antony	15215071	58/ F	W	84	150/80	E4V4M6	C	I	F	1	NA	A	N	+	+	N	156	A	28	0.8	159 /14	128/ 62	A	P	M
15	Verumanohar	15219301	60/ M	W	92	180/90	E4V4M6	C	I	F	3	NA	A	N	+	+	N	138	P	16	0.6	194 /15	136/ 63	P	P	M
16	Nesayyan	15219368	75/ M	W	74	170/90	E4V4M4	C	NI		2	NA	A	N	+	+	N	113	P	15	0.8	168 /10	134/ 68	P	P	P
17	Omana	15217737	59/ F	W	78	160/100	E4V4M5	C	I	F	0	NA	A	N	+	+	N	100	P	18	0.8	177 /13	146/ 58	A	P	M
18	Krishna	15225906	70/ F	W	78	170/100	E4V4M5	C	I	F	3	NA	A	N	+	+	N	236	A	27	0.6	180 /13	137/ 64	P	P	M
19	Selvan	1522978	45/ M	W	56	200/100	E4V4M4	C	NI		1	NA	A	N	+	+	N	140	P	25	0.7	154 /12	154/ 45	P	P	P
20	Suja	1528570	56/ F	W	80	180/110	E4V4M6	C	NI		0	NA	A	N	+	+	N	134	P	31	0.7	168 /14	127/ 56	A	P	A

MASTER CHART

S.No.	Name	IP No.	Age/sex	Presenting Complaints	Pulse(bpm)	BP mmHg	GCS	HMF conscious	CN involvement	Which nerve	Motor system	Sensory system	Meningeal involvement	Skull & spinal cord	CVS (S1S2)	RS (B/ NVBS)	Abdomen	RBS(mg/dl)	Urine microalbumin	Urea (mg/dl)	Creatinine (mg/dl)	TC/TG	LDPL/HDL	ECG(LVH)	CTbrain infarct	Territory
21	Southami	15243577	74/F	W	84	140/80	E4V4M6	C	I	F	4	NA	A	N	+	+	N	180	P	38	1	164/12	146/61	A	P	M
22	Kamalakshi	16008511	72/F	W	88	180/90	E4V4M4	C	I	F	2	NA	A	N	+	+	N	98	P	32	1	184/15	143/60	P	P	M
23	Rosely	16015127	54/F	W	78	170/100	E4V4M6	C	I	F	3	NA	A	N	+	+	N	256	P	27	0.8	166/14	138/70	A	P	M
24	Dhasamma	14153309	80/F	W	92	180/90	E4V4M5	C	I	F	2	NA	A	N	+	+	N	136	P	11	0.6	206/15	144/56	P	P	M
25	Naja Rajan	16038608	72/M	W	78	140/80	E4V4M4	C	I	F	3	NA	A	N	+	+	N	326	A	35	1.2	189/10	152/64	A	P	M
26	K.T. Thambi	16057784	65/M	W	76	150/90	E4V4M6	C	NI		1	NA	A	N	+	+	N	130	P	33	1.2	168/99	158/48	A	P	A
27	Arunachalam	16060194	70/M	W	72	160/80	E4V4M6	C	NI		3	NA	A	N	+	+	N	98	P	23	1.3	185/10	142/78	A	P	P
28	Benedict	16068219	53/M	W	82	160/90	E4V4M6	C	I	F	4	NA	A	N	+	+	N	156	A	17	0.8	129/13	138/78	A	P	M
29	Saraswathy	235884	76/F	W	68	180/100	E4V4M4	C	NI		4	NA	A	N	+	+	N	106	P	16	0.6	165/14	138/64	A	P	P
30	Saradha	236843	67/F	W	66	170/100	E4V4M6	C	I	F	0	NA	A	N	+	+	N	200	P	27	1.2	166/10	146/55	P	P	M
31	Krishnan	236247	64/M	W	50	200/100	E4V4M6	C	I	F	3	NA	A	N	+	+	N	90	P	33	0.6	178/13	142/65	A	P	M
32	Thangayya	237788	60/M	W	86	180/90	E4V4M5	C	I	F	2	NA	A	N	+	+	N	156	P	40	0.8	201/14	137/45	P	P	M
33	Rosamma	237794	67/F	W	72	180/100	E4V4M5	C	NI		3	NA	A	N	+	+	N	180	P	33	0.8	167/98	148/63	A	P	A
34	Kumaresan	241282	58/M	W	62	180/110	E4V4M6	C	NI		0	NA	A	N	+	+	N	128	P	21	0.7	199/13	154/65	A	P	A
35	Kasthuri	241378	55/F	W	60	200/110	E4V4M6	C	I	F	1	NA	A	N	+	+	N	132	P	37	1.1	189/14	137/45	P	P	M
36	Jancybai		57/F	W	68	180/90	E4V4M5	C	I	F	4	NA	A	N	+	+	N	150	P	22	1.1	167/94	158/66	P	P	M
37	Gopala Krishnan		65/M	W	74	180/90	E4V4M6	C	I	F	2	NA	A	N	+	+	N	111	P	31	0.8	164/13	138/65	P	P	M
38	Parameshwaran	244640	85/M	W	72	150/90	E4V4M4	C	NI		1	NA	A	N	+	+	N	212	P	21	0.7	207/13	124/64	A	P	P
39	Selva	245486	51/M	W	78	130/80	E4V4M5	C	I	F	4	NA	A	N	+	+	N	98	A	18	0.9	198/13	141/67	A	P	M
40	Rajammal	246070	68/F	W	76	130/90	E4V4M6	C	I	F	2	NA	A	N	+	+	N	166	A	16	1.3	184/14	138/86	A	P	M
MASTER CHART																										

S.No.	Name	IP No.	Age/sex	Presenting Complaints	Pulse(bpm)	BP mmHg	GCS	conscious	HMF involvement	CN	Which nerve	Motor system	Sensory system	Meningeal involvement	Skull & spinal cord	CVS (S1S2)	RS (B/l NVBS)	Abdomen	RBS(mg/dl)	Urine microalbumin	Urea (mg/dl)	Creatinine (mg/dl)	TC/TG	LDPL/HDL	ECG(LVH)	CTbrain infarct	Territory
41	Saraswathy	247422	83 /F	W	86	180/ 90	E4V4M6	C	I	F	3	NA	A	N	+	+	N	150	P	20	1.1	157/ 92	125 /56	P	P	M
42	Sundaran	249646	76 /M	W	78	140/ 80	E4V4M6	C	NI		3	NA	A	N	+	+	N	134	A	08	1	168/ 99	142 /66	A	P	A
43	Pakiyam	251430	63 /F	W	66	200/ 90	E4V4M6	C	NI		0	NA	A	N	+	+	N	178	P	22	0.9	159/ 138	128 /56	A	P	P
44	Anikuttan	1600426	40 /M	W	64	190/ 100	E4V4M6	C	NI		4	NA	A	N	+	+	N	94	P	27	1.1	146/ 102	134 /58	A	P	P
45	Balu	1604871	72 /M	W	74	170/ 90	E4V4M5	C	NI		4	NA	A	N	+	+	N	144	P	16	1	187/ 155	124 /56	P	P	P
46	Kamulan	1605106	70 /M	W	66	140/ 80	E4V4M6	C	I	F	1	NA	A	N	+	+	N	107	A	33	0.8	164/ 123	154 /68	A	P	M
47	Gosala	1608677	61 /F	W	56	190/ 110	E4V4M6	C	I	F	3	NA	A	N	+	+	N	134	P	32	0.9	155/ 111	134 /53	A	P	M
48	Omana	232470	75 /F	W	60	200/ 100	E4V4M5	C	I	F	0	NA	A	N	+	+	N	265	P	20	1.3	206/ 164	121 /72	P	P	M
49	Vairamuthu	235898	53 /M	W	78	180/ 100	E4V4M6	C	NI		3	NA	A	N	+	+	N	93	P	35	1.2	182/ 134	132 /55	A	P	A
50	Sebulone	236477	75 /M	W	76	170/ 100	E4V4M6	C	I	F	0	NA	A	N	+	+	N	207	P	37	0.8	168/ 144	145 /67	A	P	M

MASTER CHART

KEY TO MASTER CHART

S. No: Serial number

IP no: Inpatient number

Sex: M- Male

F – Female

Presenting Complaints: W - Weakness

Pulse: Bpm – Beats per minute

BP: Blood Pressure

GCS – Glassgow coma scale

E – Eye opening

V – Verbal response

M – Motor response

HMF – Higher Mental Function

- C - Conscious

CN involvement – Cranial nerve involvement

- I – involved
- NI – not involved

Facial – Facial nerve involvement

Motor System – Power

- 0 – No response
- 1 – Flickering movement
- 2 – Movement with gravity eliminated
- 3 – Movement against gravity
- 4 – Movement against resistance
- 5- Normal movement

Sensory System: NA – Not affected

Meningeal involvement: A – Absent

Skull & spinal cord: N – Normal

CVS – Cardiovascular System

S_1 – First heart sound

S_2 – Second heart sound

RS – Respiratory system

B/L NVBS – Bilateral Normal Vesicular Breath Sound

Abdomen: N - Normal

RBS – Random Blood Sugar

Urine microalbumin: 1. P – Present

2. A - Absent

TC – Total cholesterol

TG – Triglycerides

LDL – Low density Lipoprotein

HDL – High density Lipoprotein

ECG – Electrocardiography

- LVH – Left ventricular hypertrophy
- P – Present
- A - Absent

CT Brain infarct: P - Present

Territory: M – Middle cerebral artery

P – Posterior cerebral artery

A – Anterior cerebral artery